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EXPERIMENTAL STUDY

The Role of Executive Functioning in Spontaneous Confabulation

Gudrun M. S. Nys, MSc, Martine J. E. van Zandvoort, PhD,*† Gerwin Roks, MD, PhD,‡ L. Jaap Kappelle, MD, PhD,† Paul L. M. de Kort, MD, PhD,‡ and Edward H. F. de Haan, PhD*†*

Objective: To follow the recovery course of a patient who exhibited an amnesic-confabulatory syndrome in conjunction with severe executive dysfunction in the first week following bithalamic infarction.

Background: Previous studies have shown that spontaneous confabulation originates from the combination of amnesia and executive dysfunction and that the degree of confabulation is determined by the degree of executive dysfunction. However, a few studies have also reported a dissociation between spontaneous confabulation and executive dysfunction. Therefore, the role of executive functioning in spontaneous confabulation is presently unclear.

Method: Clinical examinations, magnetic resonance imaging (MRI), and cognitive and behavioral assessments with a focus on executive functions were conducted within the first week poststroke and after 6 months.

Results: MRI showed a bithalamic infarction involving the territory of the paramedian arteries predominantly affecting the dorsomedial and intralaminar nuclei of the thalami. Disappearance of spontaneous confabulation paralleled a specific recovery in mental flexibility, whereas all other executive components and long-term memory remained severely impaired at 6 months poststroke.

Conclusions: Our case study provides additional evidence that mental flexibility, but not executive functioning in general, is a prerequisite for spontaneous confabulation. Direct or indirect functional deactivation of dorsolateral prefrontal cortex may be necessary for the development of spontaneous confabulation.

Key Words: mental flexibility, executive functions, confabulation, amnesia, recovery

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The thalamus plays an important role as a relay center that is connected with multiple brain regions such as the prefrontal cortex, the brainstem, the cerebellum, limbic and paralimbic areas, diencephalic areas, and the basal ganglia.[1,2] Four arteries are responsible for the blood supply to the thalamus in each hemisphere: (1) the polar artery of Percheron supplies the anterior part of the thalamus, (2) the thalamosubthalamic paramedian artery supplies the medial structures, (3) the thalamogeniculate artery of Foix and Hillemand supplies the posterior part, and (4) the posterior choroidal arteries supply the ventral regions of the thalamus.[3,4] One single artery may supply thalamic territories on both sides, and therefore, bithalamic infarctions can occur.[2,3] Patients suffering from acute bithalamic infarction often demonstrate a variety of marked neurologic and neuropsychological symptoms depending on which territory of the thalami is involved. Patients with infarctions involving the territory of the paramedian artery typically demonstrate the most severe clinical picture.[2,3] Neurologic symptoms in these patients include disorders of consciousness, sleep disorders, vertical gaze paresis and convergence disorders, motor weakness, and ataxia.[4] The most prominent neuropsychological disturbances in the first period after the stroke are global amnesia[2,4–8] and abulia.[9,10] Other frequently reported cognitive impairments are confabulations,[7,9,11] impairments in executive functioning,[12,13] verbal reasoning, and mild language disturbances.[7,11] Although amnesia and abulia typically remain chronic in these patients, verbal reasoning and language capacities substantially recover in most reported cases.[6,7,14] Prognosis with respect to confabulations or executive impairments is less clear. Also, the neuropsychological correlates of spontaneous confabulation are still largely debated. Some authors argue that spontaneous confabulation originates from the combination of amnesia and executive dysfunction.[15–17] However, there is also evidence of a dissociation between executive functioning and spontaneous confabulation in amnesic patients.[18–21] An important explanation for these contrasting findings may lay in the fact that "dysexecutive functioning" is a very general and ill-defined concept. It is considered to be "a product of the coordinated operation of various processes to accomplish a particular goal in a flexible manner."[22, p 147] Several mechanisms or systems responsible for the coordinated operation of these various processes have been proposed, for example, the "executive control system,"[22,23] the "central executive,"[24] the "executive-attention framework,"[25] or the "supervisory system."[26] The functions under this executive control system are called the "executive functions," which consist of a range

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*Psychological Laboratory, Helmholtz Institute, Utrecht University, Utrecht, the Netherlands; †Department of Neurology, University Medical Centre, Utrecht, the Netherlands; and ‡Department of Neurology, St. Elisabeth & Tweesteden Hospital, Tilburg, the Netherlands.
Supported by grant no. 2000.23 from the Netherlands Heart Foundation.
Reprints: G. M. S. Nys, Psychological Laboratory, Utrecht University, Heidelberglaan 2, 3584 CS Utrecht, the Netherlands (e-mail: g.nys@fss.uu.nl).

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of dissociable subcomponents such as planning, response suppression, and mental flexibility.[27–29] Tasks tapping these executive functions activate not only prefrontal regions in the brain but also subcortical structures (eg, striatal structures and thalamus) and cerebellar areas.[27,30] A methodological issue is that up to now, most studies have focused on assessing executive functioning using only a limited number of executive neuropsychological tasks, while subsequently generalizing their results to all aspects of executive functioning. In our opinion, it is important to scrutinize the role of the different aspects of executive functioning in relation to confabulation.

Furthermore, in most studies no distinction is made between "spontaneous" confabulations and "provoked" confabulations, which is a dichotomy originally proposed by Kopelman in 1987.[31] However, there have been a number of studies showing a double dissociation between these two types of confabulations,[32–33] indicating that they represent two distinct disorders rather than different degrees of the same disorder as suggested previously.[15,34,35] Spontaneous confabulations are the most striking in appearance and are characterized by the absolute certainty with which they are uttered in conversation. Occasionally, spontaneous confabulators act according to their false beliefs, and even though spontaneous confabulations sometimes tend to have a bizarre content, they are mostly based on true events or habits in the past.[35] In contrast, provoked confabulations occur more frequently, can be elicited by direct questioning,[16] and may reflect a normal response to a faulty memory.[31] Moreover, they can be can be induced in healthy controls when they are forced to retrieve details from an imprecise memory.[19] To study the exact relationship between confabulation and executive functioning, therefore, it seems important to fractionate these concepts.

In this single case report, we demonstrate a patient with bithalamic infarction in the territory of the paramedian thalamosubthalamic artery, causing an amnesic-confabulatory syndrome in conjunction with severe executive dysfunction. We followed the course of these neuropsychological deficits over a 6-month period.

CASE DESCRIPTION

LW is a 46-year-old, left-handed clerk with 16 years of education and an unremarkable medical history. One morning, he did not show up at work. His parents found him at home in a state of total confusion. Subsequently, he was brought to the hospital, where he demonstrated a normal consciousness, a disturbed orientation, a severely affected memory, marked perseveration, confabulation, associative thinking, and anosognosia. Besides a slightly ataxic walking pattern, there were no focal neurologic deficits. His blood pressure was within normal limits. CT of the brain, ECG, CSF, and laboratory studies aimed at glucose levels, renal function, autoimmune diseases, dyslipidemia, and clotting disorders showed no abnormalities. EEG showed marked slow activity, predominantly in frontal, prefrontal, and basal temporal areas. At 3 days poststroke, MRI revealed a bithalamic infarction in the territory of the paramedian arteries predominantly affecting the dorsomedial and intralaminar nuclei of the thalami; at 6 months poststroke, the residual lesion was considerably smaller on the right side than on the left side (Fig. 1). Extensive laboratory studies, cardiologic screening including a transesophageal echocardiogram, duplex of the carotid arteries, and cerebral angiography revealed no cause for the infarction.

METHODS

Neuropsychological examination was performed on day 8 and at 6 months poststroke (Table 1). The assessments included the following tests:

Estimated premorbid verbal IQ: measured with the National Adult Reading Test (Dutch Version).

Working memory: Nonverbal and verbal working memory were assessed, respectively, by means of the Corsi block span and the WAIS-III digit span.

Memory: Verbal memory was evaluated by means of the Rey Auditory-Verbal Learning test (Dutch Version), and nonverbal memory by means of the Rey-Osterrieth Complex Figure. Two subtests of the Wechsler Memory

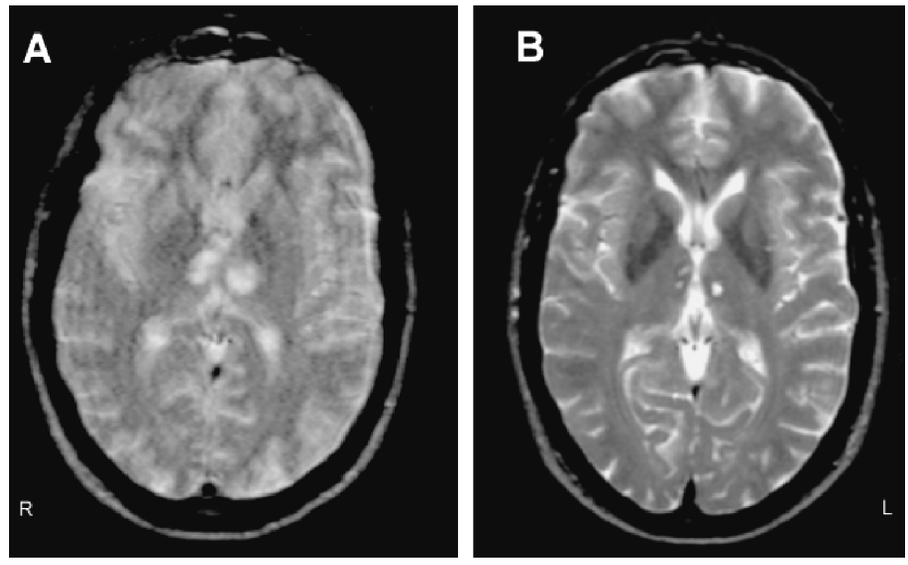

FIGURE 1. Axial T2-weighted MRI showing bilateral thalamic infarction in the paramedian thalamosubthalamic territory on day 3 post-stroke (A) and at 6 months post-stroke (B).

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TABLE 1. Neuropsychological Test Results

	February 2002		August 2002	
Working memory				
Digit span	7	Impaired	10	Impaired
Corsi block span	5	Average	5	Average
Long-term memory				
Rey Auditory Verbal Learning Test				
Immediate recall	7-4-4-3-0/15	Impaired	7-7-8-5-6/15	Borderline
Delayed recall	0/15	Impaired	0/15	Impaired
Passive recognition	18/30	Impaired	25/30	Impaired
Rey-Osterrieth Complex Figure				
Copy	35/36	High average	36/36	Superior
Delayed Recall	0/36	Impaired	0/36	Impaired
Wechsler Memory Scale				
Paragraph recall	ND			
Immediate recall		–	6/22	Impaired
Delayed recall		–	0/22	Impaired
Visual reproduction	ND			
Immediate recall		–	17/41	Impaired
Delayed recall		–	0/41	Impaired
Executive functioning				
Response suppression				
Stroop test	140.5s (3 errors)	Impaired	120.2s (0 errors)	Impaired
Word generation				
Letter fluency (NA)	0	Impaired	5 (2 + 3)	Impaired
Planning				
Zoo test (BADS)	0	Impaired	0	Impaired
Mental flexibility				
Brixton Anticipation test	47 errors	Impaired	13 errors	>Average
Visual Elevator (TEA)	0/10	Impaired	9/10	>Average
	time/switch ND	–	time/switch = 4.27	<Average

ND, not determined; BADS, Behavioral Assessment of the Dysexecutive Syndrome; TEA, Test of Everyday Attention.

Scale-Revised, Logical Memory and Visual Reproduction, were added in the follow-up evaluation.

Executive functioning: Four different aspects of executive functioning were evaluated: (1) *resistance to interference* was evaluated by means of the Stroop Color and Word test; (2) *planning abilities* were administered by means of the Zoo test (BADS); (3) *mental flexibility* was administered with two tasks—the Visual Elevator (TEA)[36] and the Brixton Spatial Anticipation task; (4) *word generation* was measured with letter fluency (N and A; each 1 minute).

RESULTS

Results of both neuropsychological examinations are summarized in Table 1.

Early Neuropsychological Assessment

During his 2-month convalescence at the hospital, LW frequently showed both spontaneous and provoked confabulations. In spontaneous conversation, for example, he said he was living in a sect, although he was actually living alone. During neuropsychological examination, he thought he was participating in a television quiz with the neuropsychologist as the quizmaster, and he saw an audience sitting beside the quizmaster, indicating that he showed visual hallucinations congruent with his confabulations. Finally, he often acted on his confabulations (eg, occasionally he left his bed to wander around the ward, thinking he was working at the hospital as a physician).

LW was disoriented in person, time, and place. His premorbid verbal IQ was estimated at 118 (NART). Verbal working memory was impaired, and nonverbal working memory was average. LW demonstrated intact visual fields on confrontation testing, suggesting that his visual hallucinations were not primarily caused by visual sensory impairments. In addition, he was unimpaired in visual construction; that is, LW's copy of the Complex Figure of Rey was entirely within normal limits. He suffered from a severe learning deficit with both verbal and visual material. He showed an abnormal learning curve for verbal material, characterized by a steep decrease in performance over trials. On verbal recognition, he reported 3/15 false negatives and 9/15 false positives, indicative of false recognition. He was not able to recall any task, even after a delay of 5 minutes, which is characteristic of a severe anterograde amnesia. In addition, LW had a retrograde

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amnesia for information extending up to 5–6 years. He was convinced that he was still in his previous employment and was shocked to hear that family members had died during the last couple of years. Furthermore, he had severe impairments on all components of executive functioning. During the examination he was disinhibited and frequently showed mood swings from euphoria to extreme apathy. He demonstrated severe perseveration during conversation and showed utilization behavior (eg, opening all closets when entering the test room or drawing when a pen was in his surroundings irrespectively of the task he was doing). He demonstrated no insight into his condition. Unless encouraged, he did not initiate any goal-directed activity (abulia).

Follow-up Assessment

At 6 months poststroke, the spontaneous confabulations had abated completely. LW still demonstrated confabulations on direct questioning, but this time his responses were plausible instead of bizarre. For example, when asked what he had for dinner the day before, he said he made himself a stew at home. In contrast to the marked certainty about the veracity of his answers in the acute phase, he was now more insecure about his responses. His brother indicated that the answer was wrong and that LW always had dinner at his parents' place.

Tests for immediate verbal memory showed a slight improvement. Verbal recognition improved, mainly through a decrease in false recognition. Retrograde amnesia was still markedly present but had shrunk to a period of 2–3 years. Interestingly, a remarkable recovery in executive functioning was found with respect to two tests, the Brixton Spatial Anticipation Test and the Visual Elevator. The Visual Elevator is a measure of attentional switching,[36] whereas the Brixton is a measure of strategic switching and problem solving.[37] Both of these tests have in common that they tap one specific aspect of executive functioning, that is, mental flexibility, which is the ability to shift behavior readily to conform to rapidly changing demands dictated by the environment.[29] Apparently, LW now had regained the capacity to shift strategy or set when necessary, and he was able to do this at a reasonable speed. However, he was still severely impaired on all other executive components, that is, planning, response suppression, and word generation. These impairments were also apparent in his daily behavior, in which he was disorganized, abulic, and disinhibited. Although his sickness insight had improved to some extent, he still was not fully aware of his impairments.

DISCUSSION

Spontaneous confabulation has been reported in a variety of neurologic patients, such as patients with bilateral infarction in the territory of the paramedian artery,[4,7,11] and also in Wernicke-Korsakoff patients as well as patients with hypothalamic damage, traumatic brain injury, multiple sclerosis, herpes simplex encephalitis, ruptured anterior communicating artery aneurysms, and dementia.[35] These patients typically have in common the presence of both amnesia and executive disorders. Therefore, it has been argued that spontaneous confabulation originates from the combination of amnesia and executive dysfunction[15–17] and that the degree of confabulation is determined by the degree of executive dysfunction.[16] Nevertheless, a few studies have reported a dissociation between spontaneous confabulation and executive dysfunction[20,21] or did not find differences in executive performance between spontaneous confabulators and nonconfabulators.[19,20] In these studies, however, executive functioning was not extensively studied, and hence, there might be a dissociation in executive components that are related to spontaneous confabulation as has been previously suggested.[16,38,39] Based on the pattern of recovery in LW, we want to stress the role of one specific executive component. Disappearance in spontaneous confabulation was accompanied by a recovery on two executive tasks that have in common that they tap mental flexibility, whereas all the other executive components assessed in this study (that is, planning, word generation, and response suppression) remained severely impaired at 6 months poststroke. These findings suggest that mental flexibility in particular is associated with spontaneous confabulation. Mental flexibility involves flexibly shifting "from one mental state, directed toward a particular reaction tendency, to another."[40, p 80] More concretely, this ability allows a person to switch to a new strategy when a certain routine strategy has become unproductive. Inflexibility results in perseveration,[27] in which patients seem magnetically attracted to whatever is in their perceptual field or current thought. The next question is how perseveration and spontaneous confabulation are related to each other. A possible underlying mechanism is that, when irrelevant thoughts or percepts intrude into the ongoing reality or an ongoing strategy because of a spatial or temporal contiguity, it becomes impossible for these patients to switch, on the one hand, between realities, resulting in confabulation, or, on the other hand, between strategies, resulting in inflexibility. This is, however, a speculative account, and this issue awaits further research.

Convergent evidence for the association between mental flexibility and confabulation comes from studies on persistent spontaneous confabulation. Patients showing long-term confabulation typically demonstrate severe impairments in mental flexibility.[41,42] Moreover, a study by Burgess and co-workers[28] pointed out that the degree of perseveration as estimated with the Dysexecutive Questionnaire (BADS)[43] loaded on the same factor as the degree of confabulation, which is also suggestive of a close relationship between these impairments. Furthermore, a recent group study investigated the clinical course and lesion characteristics of spontaneous confabulators.[18] Patients with orbitofrontal and basal forebrain lesions demonstrated the fastest recovery from spontaneous confabulation. Only one patient continued to confabulate for more than 3 years, and his lesion interrupted thalamic connections to dorsolateral prefrontal cortex. This is also the region that has been shown to be critically involved in mental flexibility in a large number of functional imaging studies and lesion studies.[40,44–48] Therefore, direct or indirect functional deactivation of dorsolateral prefrontal cortex might be a prerequisite for the occurrence of spontaneous confabulation in amnesic patients.

LW's initial scan showed fairly extensive bilateral thalamic lesions in the mediodorsal and intralaminar nuclei. A PET study in patients with similar brain lesions has demonstrated that the severe cognitive impairments in these patients are probably caused by hypoperfusion in diffuse bilateral

cortical areas through interruption of extensive thalamocortical connections, mainly to prefrontal, temporal, and posterior parietal regions.[1] Because subcortical strokes are often followed by a substantial recovery, perhaps through changes in cortical function[49] and/or reorganization of the thalamus itself,[50] LW's recovery might have occurred through reactivation of prefrontal areas involved in both mental flexibility and spontaneous confabulation. However, additional research with neuroimaging of hypoperfused regions at the same time as detailed cognitive testing is needed to delineate the exact brain structures involved in spontaneous confabulation.

Finally, we want to report on another interesting and unusual finding. Whereas most patients, including patients with amnesia, typically show an improvement in performance with each subsequent trial, LW showed an unusual verbal learning curve in which his performance deteriorated over trials. This deficit has only recently been reported by Heilman and Adams,[51] who introduced the term *cognitive impersistence* to refer to this within-session decline of performance. These authors could not find any prior references to this phenomenon. However, Stuss and co-workers have reported on this phenomenon in 1988, where they introduced a patient, RC, who showed the same decline in performance over trials.[7] Interestingly, RC also sustained a bilateral thalamic infarction, similar to our patient's lesion. No ready explanation has been proposed so far for the rapid loss of information in these patients. Further research and case reports are needed to clarify the nature of this disorder.

To conclude, our case study sheds new light on the ongoing controversy about whether or not impairment in executive functioning is required in spontaneous confabulation. It suggests that mental flexibility, but not executive functioning in general, is related to spontaneous confabulation.

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