

Clinical Note

Disrupted auto-activation, dysexecutive and confabulating syndrome following bilateral thalamic and right putaminal stroke

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Abstract. *Objective:* Clinical, neuropsychological, structural and functional neuroimaging results are reported in a patient who developed a unique combination of symptoms after a bi-thalamic and right putaminal stroke. The symptoms consisted of dysexecutive disturbances associated with confabulating behavior and auto-activation deficits.

Background: Basal ganglia and thalamic lesions may result in a variety of motor, sensory, neuropsychological and behavioral syndromes. However, the combination of a dysexecutive syndrome complicated at the behavioral level with an auto-activation and confabulatory syndrome has never been reported.

Methods: Besides clinical and neuroradiological investigations, an extensive set of standardized neuropsychological tests was carried out.

Results: In the post-acute phase of the stroke, a dysexecutive syndrome was found in association with confabulating behavior and auto-activation deficits. MRI showed focal destruction of both thalami and the right putamen. Quantified ECD SPECT revealed bilateral hypoperfusions in the basal ganglia and thalamus but no perfusion deficits were found at the cortical level.

Conclusion: The combination of disrupted auto-activation, dysexecutive and confabulating syndrome in a single patient following isolated subcortical damage renders this case exceptional. Although these findings do not reveal a functional disruption of the striato-ventral pallidal-thalamic-frontomesial limbic circuitry, they add to the understanding of the functional role of the basal ganglia in cognitive and behavioral syndromes.

Keywords: Putamen, bithalamic stroke, confabulatory syndrome, dysexecutive syndrome, psychic akinesia

1. Introduction

Ischemic lesions of the basal ganglia and the thalamus may result in clinical syndromes characterized by

a wide range of symptoms varying from pure motor deficits to complex sensory, neuropsychological and behavioral alterations [5,52,67]. There is ample evidence that ischemic lesions of the basal ganglia result in motor and neurobehavioral syndromes in which dystonia and apathy are common [6,14,21,28,32,52,54,71]. Bilateral vascular thalamic lesions may induce a wide range of neurobehavioral symptoms such as: 1) transient vigilance disorders, 2) hypersomnia, 3) verti-

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cal gaze palsy, 4) anterograde amnesia, 5) speech and language disturbances and 6) apathy [3,20,24,30,31,35,38,49,50,57,59,61,73]. A typical syndrome of apathy which has been documented in association with uni- or bilateral lesions of the thalamus and/or the basal ganglia [10–12,14,15,19,23,31,68,70,72] is known as ‘athymhormia’, ‘psychic akinesia’, or ‘loss of psychic self-activation’ (LPSA), of which two distinct subtypes can be distinguished. The first variant is a condition of extreme loss of activity and motivation resulting in apathy, motor and verbal asponaneity with subjacent ‘mental emptiness’. Remarkable in this condition is the sharp contrast between impaired self-activation and intact heteroactivation of behavior. The second behavioral condition consists of an apparent flatness or poor expressiveness of affect and the disappearance of psychic life [11,13,23,39,42,45].

Levy and Dubois [62] define apathy as an ‘observable pathologic behavioral syndrome consisting of a quantitative reduction of self-generated voluntary and purposeful behaviors’ and distinguish three subtypes in the underlying mechanisms of apathy: ‘emotional-affective’, ‘cognitive’ and ‘auto-activation’. Apathy related to a disruption of ‘emotional-affective’ processing refers to a reduction of goal-directed behavior due to an inability to associate affective and emotional signals with ongoing and forthcoming behaviors, whereas apathy related to a disruption of ‘cognitive’ processing is related to cognitive deficits in elaborating plans of action. The third and most severe form of apathy consists of a disruption of auto-activation processing which refers to the inability of self-activating and self-initiating thoughts and activities: this affects both cognitive and emotional responses. This behavioral syndrome is similar to ‘athymhormia’, ‘psychic akinesia’ and ‘loss of psychic self-activation’. The ‘auto-activation’ deficit mostly involves bilateral lesions of the thalamus, the pallidal globe, the caudate nucleus or the striato-pallidal region. Although the condition most commonly occurs after bilateral lesions, patients with unilateral lesions have been reported [1,4,8,11,23,26,29,31,33,37,45,46,51,53,65,66,68,69,71]. Besides psychic akinesia, impairments in executive functioning and confabulations are also associated with bi-thalamic lesions [20,22,38,40,70,73].

This article reports the clinical, neuropsychological, structural and functional neuroimaging results in a patient who developed a unique set of symptoms consisting of a dysexecutive syndrome in association with confabulating behavior and auto-activation deficits after a bi-thalamic and right putaminal stroke.

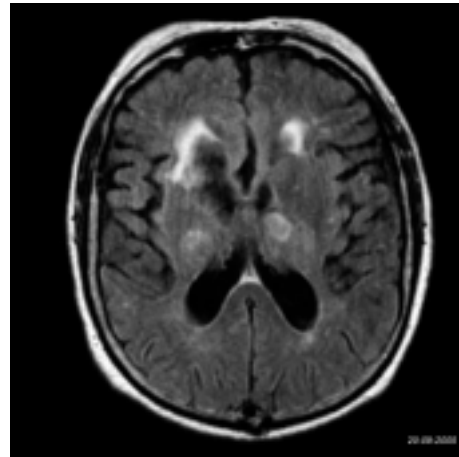


Fig. 1. Brain MRI axial FLAIR carried out five days after stroke revealing focal ischemic damage to both thalami and the right putamen.

2. Case report

A 78-year-old right-handed woman was admitted to hospital after a sudden onset of incoherent speech and confusion. Medical history was unremarkable. Apart from mild disorientation, clinical neurological examination on admission was normal. Tests of eye movements and other cranial nerve functions were normal. Brain magnetic resonance imaging (MRI) in the acute phase (5 days post-onset) revealed ischemic lesions in both thalami and the right putamen (Fig. 1). Comparison of the MRI-images with an atlas of neuro-imaging and neuroanatomy [27] showed unilateral involvement of the left medial and anterior thalamic nucleus, unilateral involvement of the right dorsal anterior nucleus, bilateral involvement of the intralaminar nuclei, unilateral involvement of the right putamen and bilateral involvement of the frontal subcortical white matter.

In the post-acute phase of the stroke (2 weeks post-onset), anomie language disturbances, concentration and memory deficits were found in combination with a dysexecutive syndrome. At the behavioral level, a combination of inhibitory and disinhibitory symptoms were found. Seven months post-onset, repeat neurolinguistic and neuropsychological investigations revealed severe frontal dysexecutive dysfunctions in combination with a unique neurobehavioral syndrome characterized by a severe loss of psychic self-activation and the presence of a confabulatory syndrome. ^{99m}Tc ECD single photon emission computed tomography (SPECT) scan was carried out eight months post-onset. Trans-axial images with a pixel size of 3.56 mm were anatomically standardized using SPM and compared to a stan-

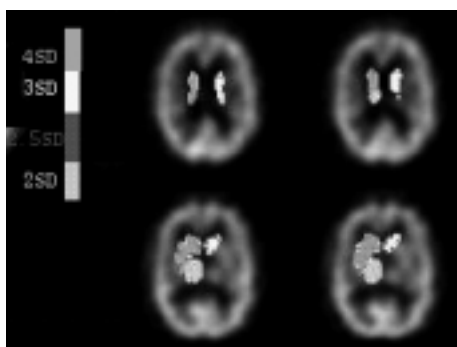


Fig. 2. Quantified ECD SPECT eight months after stroke disclosing hypoperfusions in the thalamus, both caudate nuclei and the left lentiform nucleus.

standard normal and SD image obtained from 15 normal ECD perfusion studies. Using a 31 ROI template the z-scores (SD) were then calculated for each region. A regional z-score of > 2.0 was considered significant. In comparison to normal database findings the quantified baseline ECD SPECT at eight months post-stroke showed a significant decrease of perfusion in both thalami (left: -1.96 SD; right: -2.47 SD), both caudate nuclei (left: -5.24 SD; right: -3.92 SD) and the left lentiform nucleus (-4.97 SD). No deficits were found at the cortical level (Fig. 2).

3. Methods

Formal neuropsychological investigations were carried out two weeks (acute phase) and seven months (late phase) after the stroke. Language was assessed by means of subtests of the Boston Diagnostic Aphasia Examination (BDAE) [25], subtests of the Aachen Aphasia Test (AAT) [56], the Boston Naming Test (BNT) [18,58], subtests of the Hierarchic Dementia Scale (HDS) [41] and a semantic verbal fluency task (unpublished norms). Cognitive functions were examined by means of the Mini Mental State Examination (MMSE) [43], the Colored Progressive Matrices (CPM) [35], the Hierarchic Dementia Scale (HDS) [41], the Trail Making Test (TMT) [63,64], the Stroop Color Word Test (SCWT) [36] and the Wisconsin Card Sorting Test (WCST) [7].

4. Results

Neurolinguistic and neurocognitive test results are summarized in Table 1. In the acute phase of the stroke,

the patient obtained normal scores for auditory comprehension, written comprehension, repetition, reading and writing. Similar results were obtained in the late phase of the stroke. Two weeks post-onset, the visual confrontation naming subtest of the HDS (4/10) and the BNT (22/60; -3.91 SD) showed severe anomia. Most naming errors were semantic paraphasias (14/38) (e.g. tree for cactus), followed by perseverations (9/38), semantic neologisms (7/38) (e.g. "freezing pencil" for igloo), inadequate circumlocutions (4/38) (e.g. parts of a throw for domino) and visual errors (4/38) (e.g. cup for mask). Seven months post-onset a remission of naming errors was found (HDS: 10/10; BNT: 51/60 ($+1.41$ SD)). In a semantic verbal fluency task the patient was asked to name as many animals, means of transport, vegetables and clothes as possible within one minute for each semantic field. In the acute phase the patient produced 22 items (-1.82 SD). Her performance had improved considerably in the late phase (39 items; -0.37 SD). In the acute phase of the stroke spontaneous speech was characterized by incidences of neologistic and semantic jargon. Seven months later jargon had resolved completely. Despite normal oral language skills, the patient did not take any initiative to speak spontaneously.

At a neurocognitive level, the MMSE revealed a deficient score of 22/30 (-3.33 SD) two weeks post-onset neurological symptoms. A normalization of the total score was found seven months post-onset (26/30; -0.66 SD) [60]. As shown by the HDS subtest results, recent memory and concentration were disrupted in the acute phase. Seven months later, recent memory defects and concentration disturbances had resolved. Praxis, orientation, visual perception, gnosis and arithmetics were normal as assessed by the HDS. On the CPM, the patient obtained normal results in the acute and late phase of the stroke. Executive dysfunctions were found at both the psychometric and behavioral level. As demonstrated by a severely deficient score on the Stroop Color-Word Test (card III = percentile 1) the ability of resistance to interference in more automatic stimuli was severely disturbed in the acute phase. Seven months later performance had improved (card III = percentile 30). A severely defective result on the WCST (zero categories in 64 trials) indicated that mental flexibility, abstract reasoning, frontal planning and organization were severely impaired in both the acute and lesion phase of the stroke. Disturbed mental flexibility was also evidenced by a defective result on the TMT (part B: percentile 10).

At the behavioral level a range of frontal-like disturbances were observed such as apathy, disconcern,

Table 1
Neuropsychological test results obtained two weeks and seven months post-onset

Neuropsychological tests	Week 2	Month 7	Percentile 2/7	Max	Mean	SD
<i>Mini Mental State Examination</i>	22	26		30	27	1.5
<i>Intelligence</i>						
Raven Coloured Progressive Matrices	23	31	75/95	36		
<i>Memory</i>						
Hierarchic Dementia Scale (HDS)						
registration (item 8)	8	10		10	9.17	0.94
recent memory (item 20)	2	8		10	8.51	1.64
remote memory (item 17)	10	10		10	9.62	0.80
<i>Language</i>						
Boston Diagnostic Aphasia Examination (BDAE)						
complex auditory language comprehension	14	15	93/100	15		
Akense Aphasia Test (AAT)						
repetition total	150	150	100/100	150	144.1	8.07
Boston Naming Test (BNT)	22	51		60	43.3	5.45
Hierarchic Dementia Scale (HDS)						
naming (item 6)	4	10		10	9.87	0.34
comprehension (item 7)	10	10		10	9.85	0.42
reading (item 10)	10	10		10	9.83	0.56
writing (item 18)	10	10		10	9.06	1.26
Verbal fluency – semantic	22	39			43.4	11.76
animals	6	11		1 min		
vegetables	5	12		1 min		
clothing	5	10		1 min		
means of transport	6	6		1 min		
<i>Concentration</i>						
Hierarchic Dementia Scale (HDS)						
concentration (item 13)	7	10		10	8.94	0.87
<i>Frontal functions</i>						
Trail Making Test						
version A	–	49 sec	–/80			
version B	–	> 450 sec	–/< 10			
Stroop Color-Word Test						
card I	46 sec	36 sec	60/95			
card II	106 sec	55 sec	3/75			
card III	> 197 sec	107 sec	1/30			
Wisconsin Card Sorting Test (WCST)	0	0	0/0	64		
<i>Praxis – Orientation – Visual Perception & Gnosis Calculation</i>						
ideomotor praxis (HDS, item 3)	10	10		10	9.45	0.80
ideational praxis (HDS, item 5)	10	10		10	9.81	0.45
construction (HDS, item 12)	10	10		10	8.33	1.88
drawing (HDS, item 15)	10	10		10	8.72	0.97
orientation (HDS, item 11)	10	8		10	9.45	1.02
visual perception (HDS, item 4)	10	10		10	9.87	0.49
gnosis (HDS, item 9)	10	10		10	9.47	0.50
calculation (item 14)	10	10		10	8.83	1.26

Legend: max = maximumscore; SD = standard deviation; sec = seconds.

indifference, loss of interest, loss of critical attitude, poor motivation, flattened affect and abusive language (cursing). In addition the patient often confabulated. In spontaneous conversations, for example, she said that her husband, who died 18 years ago, did not accompany her because he dislikes hospitals. When she was asked to describe a script, such as a typical morning routine, a bizarre mix of confabulations disrupted the content of her speech. Some of these confabulations were based on true habits in the past while others were

false beliefs. Although she was a resident in a nursing home, she claimed to wake up at six o'clock to prepare breakfast for both her children. She insisted that her son lives with her on the second floor of her house and that he leaves the house every morning at seven o'clock to catch the train to go to work. She further claimed that her daughter has a room on the first floor of her house and that she leaves at eight to work in a sheltered workshop (= true but 28 years ago). The reality is that both her son and daughter had left the house a long time

ago. At the moment of the examination, she claimed that her husband stayed at home with her daughter (= false). When the examiner told the patient that she stayed in a nursing home, she said that her daughter lives with her husband now (= false).

The frontal-like inhibition disturbances and the confabulatory syndrome have remained unchanged. Her son considers the behavioral alterations as the most striking consequence of the stroke. Instead of the active, talkative woman she used to be before the stroke, she remains apathetic, passive and confabulatory. Because of these changes, she was admitted to a nursing home where she avoids contact with other residents. She hardly speaks and takes no initiative to communicate. Nevertheless, minimal levels of speech are punctuated with spontaneous and provoked confabulations. When no strong external stimulation is given, she does not do anything spontaneously. On the Middelheim Frontality Scale (MFS), she scored 7/10 [55]. The Beck Depression Inventory (BDI) did not show evidence in favor of a depressive syndrome [2].

5. Discussion

This paper reports a patient with a unique combination of cognitive and behavioral symptoms due to a bilateral thalamic and right putaminal stroke. The patient developed a dysexecutive syndrome that persisted during follow-up. In addition to impaired performance on frontal tasks evaluating mental flexibility, frontal planning and organization, loss of psychic self-activation and a confabulatory syndrome are found. Loss of psychic self-activation is characterized by apathy, loss of initiative, loss of concern, loss of critical attitude, motor and verbal asponaneity, mental emptiness and flattened affect. This behavioral syndrome shares clear semiological similarities with syndromes caused by lesions of the dorso-medial prefrontal cortex and corresponds to the third subtype of the apathetic syndrome described by Stuss et al. [16] and Levy and Dubois [62] as a 'behavioral apathetic syndrome' and an 'auto-activation deficit'. The confabulatory syndrome in this patient consists of a mix of spontaneous as well as provoked confabulations. Most impressive are the spontaneous confabulations in which the patient shows a definite certainty about the veracity of her assertions. Most of these spontaneous confabulations are based on true events or on habits in the past while the provoked confabulations mainly reflect false beliefs. These findings provide additional evidence for a positive correlation

between the degree of mental flexibility and the degree of spontaneous confabulation and are in agreement with the hypothesis of Nys et al. [22]. In this patient, a long-term coexistence of spontaneous confabulations and mental a-flexibility is observed as evidenced by the poor scores on the WCST and the TMT.

From an anatomo-clinical point of view, the persistence of executive dysfunctions may be attributed to the lesion in the medial thalamic nucleus, the intralaminar thalamic nuclei or any combination of these nuclei. Our findings are consistent with previous studies demonstrating an association between a lesion consisting of a combination of thalamic nuclei damage and a persistent dysexecutive syndrome [47–49,73]. Regarding the possible pathophysiological mechanisms underlying the neurobehavioral syndrome of psychic akinesia and/or confabulation, most research suggests disruption of the striato-ventral pallidal-thalamic-frontomesial limbic loop [9, 17,22,31,44,50,62,67]. Quantified SPECT in this patient does not support this view since no perfusion deficits were found in the anatomo-clinically suspected frontal brain regions. The lack of SPECT evidence for a functional disruption of this circuitry may indicate that bilateral involvement of the thalamus associated with putaminal damage is sufficient to induce the observed cognitive and behavioral symptoms. However, further research is necessary to clarify the role of combined lesions of the basal ganglia and thalami in dysexecutive, auto-activation and confabulatory syndromes.

6. Conclusion

A patient is reported who developed a persistent dysexecutive syndrome with confabulations and auto-activation deficits after ischemic damage to both thalami and the right putamen. There appears to be no SPECT evidence for a functional disruption of the striato-ventral pallidal-thalamic-frontomesial limbic loop. At the behavioral level the unique semiological association of the 'auto-activation deficit' and a confabulatory syndrome has – to the best of our knowledge – never been described before.

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References

- [1] A. Ali-Cherif, M.L. Royere, A. Gosset, M. Poncet, G. Salamon and R. Khalil, Troubles du comportement et de l'activité mentale après intoxication oxycarbonée. Lésions pallidales bilatérales, *Revue Neurologique* **140** (1984), 401–405.
- [2] A.T. Beck, C.H. Ward, M. Mendelson, J. Mock and J. Erbaugh, An inventory for measuring depression, *Archives of General Psychiatry* **4** (1961), 561–571.
- [3] A. Guberman and D. Stuss, The syndrome of bilateral paramedian thalamic infarction, *Neurology* **33** (1983), 540–546.
- [4] A. Lugaresi, P. Montaga, A. Morreale and R. Gallassi, Psychic akinesia following carbon monoxide poisoning, *European Neurology* **30** (1990), 167–169.
- [5] A.A. Utters and M.A. Basso, The basal ganglia: an overview of circuits and function, *Neuroscience and Biobehavioral Research* (2007), Jan Epub.
- [6] B. Dubois, B. Defontaines, B. Deweer, C. Malapani and B. Pillon, Cognitive and behavioral changes in patients with focal lesions of the basal ganglia, *Advances in Neurology* **65** (1995), 29–41.
- [7] D.A. Grant and E.A. Berg, *The Wisconsin Card Sorting Test Random Layout. Directions for scoring and administration*, University of Wisconsin: Department of Psychology, 1948.
- [8] D.I. Katz, M.P. Alexander, G.M. Seliger and D.N. Bellas, Traumatic basal ganglia hemorrhage: clinicopathologic features and outcome, *Neurology* **39** (1989), 897–904.
- [9] D. Laplane, La perte d'auto-activation psychique, *Revue Neurologique* **146** (1990), 397–404.
- [10] D. Laplane, M. Baulac, B. Pillon and I. Panayotopoulou-Achimastos, Loss of psychic self-activation. Compulsive activity of obsessional type. Bilateral lenticular lesion, *Revue Neurologique* **138** (1982), 137–141.
- [11] D. Laplane, M. Baulac, D. Widlocher and B. Dubois, Pure psychic akinesia with bilateral lesions of basal ganglia, *Journal of Neurology* **47** (1984), 377–385.
- [12] D. Laplane and B. Dubois, Auto-activation deficit: a basal ganglia related syndrome, *Movement Disorders* **16** (2001), 810–814.
- [13] D. Laplane, B. Dubois, B. Pillon and M. Baulac, Perte d'autoactivation psychique et activité mentale stéréotypée par lésion frontale, *Revue Neurologique* **144** (1988), 564–570.
- [14] D. Laplane, M. Levasseur, B. Pillon, B. Dubois, M. Baulac, B. Mazoyer, S. Tran Dinh, G. Sette, F. Danze and J.C. Baron, Obsessive-compulsive and behavioural changes with bilateral basal ganglia lesions, *Brain* **112** (1989), 699–725.
- [15] D. Laplane, D. Widlocher, B. Pillon, M. Baulac and F. Binoux, Obsessional-type compulsive behavior caused by bilateral circumscribed pallidostriatal necrosis. Encephalopathy caused by a wasp sting, *Revue Neurologique* **137** (1981), 269–276.
- [16] D.T. Stuss, R. Van Reekum and K.J. Murphy, Differentiation of stated and causes of apathy. In: *The Neuropsychology of Emotions*, Oxford: Oxford University Press, 2000.
- [17] E. Carrera and J. Bogousslavsky, The thalamus and behavior: effects of anatomically distinct strokes, *Neurology* **66** (2006), 1817–1823.
- [18] E. Kaplan, H. Goodglass and S. Weintraub, *Boston Naming Test*, USA: Lea and Febiger, 1983.
- [19] E. Kumral, D. Evyapan and K. Balkir, Acute caudate vascular lesions, *Stroke* **30** (1999), 100–108.
- [20] E. Kumral, D. Evyapan, K. Balkir and S. Kutluhan, Bilateral thalamic infarction. Clinical, etiological and MRI correlates, *Acta Neurologica Scandinavica* **103** (2001), 35–42.
- [21] E.T. Rolls, Neurophysiology and cognitive functions of the striatum, *Revue Neurologique* **150** (1994), 648–660.
- [22] G.M. Nys, M.J. Van Zwandvoort, G.M. Roks, L.J. Kappelle, P.L. De Kort and E.H. De Haan, The role of executive functioning in spontaneous confabulation, *Cognitive and Behavioral Neurology* **17** (2004), 213–218.
- [23] G. Rodier, C. Tranchant, M. Mohr and J.M. Warter, Neurobehavioural changes following bilateral infarct in the caudate nuclei: a case report with pathological analysis, *Journal of the Neurological Sciences* **126** (1994), 213–218.
- [24] H. Bewermeyer, H.A. Dreesbach, A. Rackl, M. Neveling and W.D. Heiss, Presentation of bilateral thalamic infarction on CT, MRI and PET, *Neuroradiology* **27** (1985), 414–419.
- [25] H. Goodglass and E. Kaplan, *The assessment of aphasia and related disorders*, Philadelphia: Lea and Febiger, 1983.
- [26] H.L. Klawans, R.W. Stein, C.M. Tanner and C.G. Goetz, A pure parkinsonian syndrome following acute carbon monoxide intoxication, *Archives of Neurology* **39** (1982), 302–304.
- [27] H.J. Kretschmann and W. Weinrich, *Cranial Neuroimaging and Clinical Neuroanatomy*, New York: Thieme Medical Publishers, 1992.
- [28] H. Mizuta and N. Motomura, Memory dysfunction in caudate infarction caused by Heubner's recurring artery, *Brain and Cognition* **61** (2006), 133–138.
- [29] H.A. Peters, R.L. Levine, C.G. Matthews and L.J. Chapman, Extrapyramidal and other neurologic manifestations associated with carbon disulfide fumigant exposure, *Archives of Neurology* **45** (1988), 537–540.
- [30] I. Dehaene, Bilateral thalamo-subthalamic infarction, *Acta Neurologica Belgica*, **82** (1982), 253–261.
- [31] J. Bogousslavsky, F. Regli, A. Delaloye-Bischof, G. Assal and A. Uske, Loss of psychic self-activation with bithalamic infarction: neurobehavioral, CT, MRI and SPECT correlates, *Acta Neurologica Scandinavica* **83** (1991), 309–316.
- [32] J. Brown, D. Bullock and S. Grossberg, How the basal ganglia use parallel excitatory and inhibitory learning pathways to selectively respond to unexpected rewarding cues, *Journal of Neuroscience* **19** (1999), 10502–10511.
- [33] J.K. Krauss, M. Mohadjer, A.K. Wakhloo and F. Munding, Dystonia and akinesia due to pallidoputamin lesions after disulfiram intoxication, *Movement Disorders* **6** (1991), 166–170.
- [34] J.C. Raven, J.H. Court and J. Raven, *Manual for Raven's Progressive Matrices and Vocabulary Scales*, London, 1983.
- [35] J.D. Schmahmann, Vascular syndromes of the thalamus, *Stroke* **34** (2003), 2264–2278.
- [36] J.R. Stroop, Studies of interference in serial verbal reactions, *Journal of Experimental Psychology* **18** (1935), 643–662.
- [37] L.R. Caplan, J.D. Schmahmann, C.S. Kase, E. Feldmann, G. Bacquis, J.P. Greenberg, P.B. Gorelick, C. Helgason and D.B. Hier, Caudate infarcts, *Archives of Neurology* **47** (1990), 133–143.
- [38] L. De Witte, I. Wilssens, S. Engelborghs, P.P. De Deyn and P. Mariën, Impairment of syntax and lexical semantics in a patient with bilateral paramedian thalamic infarction, *Brain and Language* **96** (2006), 69–77.
- [39] L. Milandre, M. Habib, M.L. Royere, R. Gouirand and R. Khalil, Syndrome athymorique par infarctus striato-capsulaire bilatéral, *Revue Neurologique* **151** (1995), 383–387.
- [40] M. Benabdeljil, M. El Aloui Faris, N. Kissani, S. Laaouina, M. Jiddane and T. Chkili, Neuropsychological disorders after bithalamic infarct caused by deep venous thrombosis, *Revue Neurologique* **157** (2001), 62–67.

- [41] M.G. Cole and D. Dastoor, A new hierarchic approach to the measurement of dementia, *Psychosomatics* **28** (1987), 298–305.
- [42] M. Dide and P. Guiraud, *Psychiatrie du médecin praticien*, Paris: Masson, 1922.
- [43] M.S. Folstein, S.E. Folstein and P.R. Mc Hugh, Mini-mental state : A practical method for grading the cognitive status of patients for the clinician, *Journal of Psychiatric Research* **12** (1975), 189–198.
- [44] M. Habib, Athymhormia and disorders of motivation in basal ganglia disease, *Journal of Neuropsychiatry and Clinical Neurosciences* **16** (2004), 509–524.
- [45] M. Habib, M. Poncet and Perte de l' élan vitale, de l' intérêt et de l' affectivité (syndrome athymhormique) au cours de lésions lacunaires des corps striés, *Revue Neurologique* **144** (1988), 571–577.
- [46] M.F. Mendez, N.L. Adams and K.S. Lewandowski, Neurobehavioral changes associated with caudate lesions, *Neurology* **39** (1989), 349–354.
- [47] M. Mennemeier, B. Crosson, D.J. Williamson, S.E. Nadeau, E. Fennell and E. Valenstein, Tapping, talking and the thalamus: Possible influence of the intralaminar nuclei on basal ganglia function., *Neuropsychologia* **35** (1997), 183–193.
- [48] M. Mennemeier, E. Fennell, E. Valenstein and K.M. Heilman, Contributions of the left intralaminar and medial thalamic nuclei to memory. Comparison and report of a case, *Archives of Neurology* **49** (1992), 1050–1058.
- [49] M. Radanovic, M. Azambuja, L.L. Mansur and C.S. Porto, Thalamus and language: interface with attention, memory and executive functions, *Arquivos Neuropsiquiatria* **61** (2003), 34–42.
- [50] M. Reilly, S. Connolly, J. Stack, E.A. Martin and M. Hutchinson, Bilateral paramedian thalamic infarction : a distinct but poorly recognized stroke syndrome, *Quarterly Journal of Medicine* **82** (1992), 63–70.
- [51] M. Trillet, B. Croisile, D. Tourniaire and B. Schott, Perturbations de l' activité motrice volontaire et lésions des noyaux caudés, *Revue Neurologique* **146** (1990), 338–344.
- [52] M.T. Trinidad-Herrero, C. Barcia and F. Navarra, Functional anatomy of thalamus and basal ganglia, *Children's Nervous System* **18** (2002), 386–404.
- [53] O. Godefroy, M. Rousseaux, D. Leys, P. Scheltens and J.P. Pruve, Frontal lobe dysfunction in unilateral lenticulo-striate infarcts, *Archives of Neurology* **49** (1992), 1285–1289.
- [54] P. Collins, L.S. Wilkinson, B.J. Everitt, T.W. Robbins and A.C. Roberts, The effects of dopamine depletion from the caudate nucleus of the common marmoset on tests of prefrontal cognitive function, *Behavioural Neurology* **114** (2000), 3–17.
- [55] P.P. De Deyn, S. Engelborghs, J. Saerens, J. Goeman, P. Mariën, K. Maertens, G. Nagels, J.J. Martin and B.A. Pickut, The Middelheim Frontality Score: a behavioural assessment scale that discriminates frontotemporal dementia from Alzheimer disease, *International Journal of Geriatric Psychiatry* **20** (2005), 70–79.
- [56] P. Graetz, R. De Bleser and K. Willmes, *Akense Afasietest. Handleiding*, Lisse: Swets and Zetlinger, 1990.
- [57] P. Krolak-Salmon, B. Croisile, C. Houzard, A. Setiey and P. Girard-Madoux, Total recovery after bilateral paramedian thalamic infarct, *European Neurology* **44** (2000), 216–218.
- [58] P. Mariën, E. Maempeay, A. Vervaeet, J. Saerens and P.P. De Deyn, Normative data for the Boston naming test in native Dutch-speaking Belgian elderly, *Brain and Language* **65** (1998), 447–467.
- [59] P.H.M.F. Van Domburg, H.J. Donkelaar and S.L.H. ten Notermans, Akinetic mutism with bithalamic infarction. Neurophysiological correlates, *Journal of Neurological Sciences* **38** (1996), 58–65.
- [60] R.M. Crum, I.C. Anthony, S.S. Bassett and M.F. Folstein, Population-based norms for the mini-mental state examination by age and educational level, *JAMA* **18** (1993), 2386–2391.
- [61] R. Fukatsu, T. Fuji, A. Yamadori, H. Nagasawa and Y. Sakurai, Persisting childish behavior after bilateral thalamic infarcts, *European Neurology* **37** (1997), 230–235.
- [62] R. Levy and B. Dubois, Apathy and the Functional Anatomy of the Prefrontal Cortex-Basal Ganglia Circuits, *Cerebral Cortex* **16** (2006), 916–928.
- [63] R. Reitan, Validity of the trail making test as an indication of organic brain damage, *Perceptual and Motor Skills* **8** (1958), 271–276.
- [64] R. Reitan, *Trail Making Test. Manual for Administration, Scoring and Interpretation*, Bloomington: Indiana University, 1956.
- [65] R.L. Strub, Frontal lobe syndrome in a patient with bilateral globus pallidus lesions, *Archives of Neurology* **46** (1989), 1024–1027.
- [66] R.J. Uitti, A.H. Rajput, E.M. Ashenurst and B. Rozdilsky, Cyanide-induced parkinsonism: a clinicopathologic report, *Neurology* **35** (1985), 921–925.
- [67] S. Engelborghs, P. Mariën, J.J. Martin and P.P. De Deyn, Functional anatomy, vascularisation and pathology of the human thalamus, *Acta Neurologica Belgica* **98** (1998), 252–265.
- [68] S. Engelborghs, P. Mariën, B.A. Pickut, S. Verstraeten and P.P. De Deyn, Loss of psychic self-activation after paramedian bithalamic infarction, *Stroke* **31** (2000), 1762–1766.
- [69] S.M. Pulst, T.M. Walshe and J.A. Romero, Carbon monoxide poisoning with features of Gilles de la Tourette's syndrome, *Archives of Neurology* **40** (1983), 443–444.
- [70] S. Vucic, T. Iye and R.A. Mackenzie, Neuropsychological manifestations in a case of bilateral thalamic infarction, *Journal of Clinical Neuroscience* **10** (2003), 238–242.
- [71] T. Benke, M. Delazer, L. Bartha and A. Auer, Basal ganglia lesions and the theory of fronto-subcortical loops : neuropsychological findings in two patients with left caudate lesions, *Neurocase* **9** (2003), 70–85.
- [72] T. Fukutake, K. Akada, S. Ito, T. Okuda and Y. Ueki, Severe personality changes after unilateral left paramedian thalamic infarct, *European Neurology* **47** (2002), 156–160.
- [73] Y.D. Van Der Werf, P. Scheltens, J. Linderboom, M.P. Witter, H.B. Uylings and J. Jolles, Deficits of memory, executive functioning and attention following infarction in the thalamus; a study of 22 cases with localised lesions, *Neuropsychologia* **41** (2003), 1330–1344.



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