

Case Reports

Loss of Psychic Self-Activation After Paramedian Bithalamic Infarction

S. Engelborghs, MD; P. Marien, MA; B.A. Pickut, MD, MPH;
S. Verstraeten, MA; P.P. De Deyn, MD, PhD

Background and Purpose—Loss of psychic self-activation has been described after bilateral lesions to the globus pallidus, striatum, and white matter of the frontal lobes, but it is a very rare sign of bithalamic lesions. The exact functional-anatomic mechanism underlying loss of psychic self-activation following bithalamic lesions remains to be elucidated.

Case Description—We present clinical, neuropsychological, structural, and functional neuroimaging data of an 18-month follow-up period of a man with prominent loss of psychic self-activation after coronary arteriography. Except for memory decline, accompanying symptoms remained restricted to the acute phase. The neurobehavioral syndrome consisted mainly of apathy, indifference, poor motivation, and flattened affect, and this remained unchanged during the entire follow-up period. MRI showed a bithalamic infarction involving the nucleus medialis thalami bilaterally. Single-photon emission CT revealed a severe relative hypoperfusion of both thalami, a relative hypoperfusion of both nuclei caudati, and a relative hypoperfusion mesiofrontally.

Conclusions—Single-photon emission CT data support the hypothesis that the neurobehavioral manifestations after bithalamic paramedian infarction are caused by disruption of the striatal-ventral pallidal-thalamic-frontomesial limbic loop. Probably, bilateral disruption at different levels of the striatal-ventral pallidal-thalamic-frontomesial loop may lead to a similar clinical picture consisting of loss of psychic self-activation. (*Stroke*. 2000;31:1762-1765.)

Key Words: stroke ■ thalamus ■ tomography, emission computed

Bilateral lesions of the nucleus anterior and medialis thalami can induce an amnesic syndrome characterized by deficiencies in anterograde and retrograde mnemonic functions.^{1,2} Thalamic amnesic syndromes are often accompanied by confusional states and temporary fluctuations of consciousness.³

We present a case with persistent apathy, unconcern, poor motivation, and flattened affect after recovery from acute-phase symptomatology of a bilateral paramedian thalamic infarction. The so-called loss of psychic self-activation has been described after bilateral lesions to the globus pallidus, striatum, and white matter of the frontal lobes,^{4–6} but it is a very rare sign of bithalamic infarction.⁷ Most cases with a loss of psychic self-activation after bithalamic injury have additional symptoms. Except for a memory decline, the accompanying symptoms in the present case remained restricted to the acute phase.

Case Report

During a coronary arteriography, this 58-year-old right-handed patient suddenly developed a left hemiparesis and

coma. The clinical neurological examination showed a comatose patient (Glasgow Coma Scale [GCS] E1V1M2) with equal pinpoint pupils and decerebrate rigidity. No eye movements could be elicited by the oculocephalic maneuver. Muscle tone of the left arm and leg were decreased. Tendon reflexes at the left arm and leg were increased. The left plantar response was extensor, whereas the right plantar response was flexor.

For several hours, the patient exhibited fluctuating levels of consciousness, with GCS scores varying between 4 and 14. Muscle tone and strength rapidly normalized. Pupils and eye movements, including vertical gaze, were normal. Oculocephalic testing was unremarkable. Tendon reflexes were still brisker on the left side of the body than on the right side. The left plantar response remained extensor. Consciousness was fully recovered 3 days after stroke.

The patient's behavior had dramatically changed. Instead of the active man he used to be, he had become an apathetic, passive, and indifferent person who seemed to have lost all emotional concern and initiative. His affect was clearly blunted. The patient remained indifferent to

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From the Department of Neurology and Memory Clinic (S.E., P.M., B.A.P., S.V., P.P.D.D.), Middelheim General Hospital, Antwerp, Belgium, and Department of Neurology, Laboratory of Neurochemistry and Behavior (S.E., B.A.P., P.P.D.D.), Born-Bunge Foundation, University of Antwerp, Belgium.

Correspondence to Prof Dr P.P. De Deyn, Department of Neurology and Memory Clinic, Middelheim General Hospital, Lindendreef 1, 2020 Antwerp, Belgium. E-mail ppedeyn@uia.ua.ac.be

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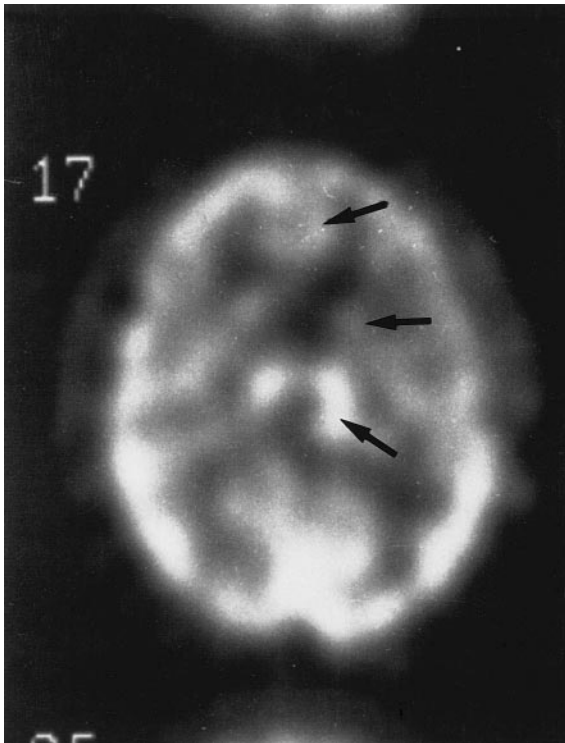


Figure 1. ^{99m}Tc -HMPAO SPECT (1 week after stroke): luxury perfusion of both thalami, relative hypoperfusion of both nuclei caudati, and relative hypoperfusion of the mesiofrontal region (all most pronounced at the left [arrows]).

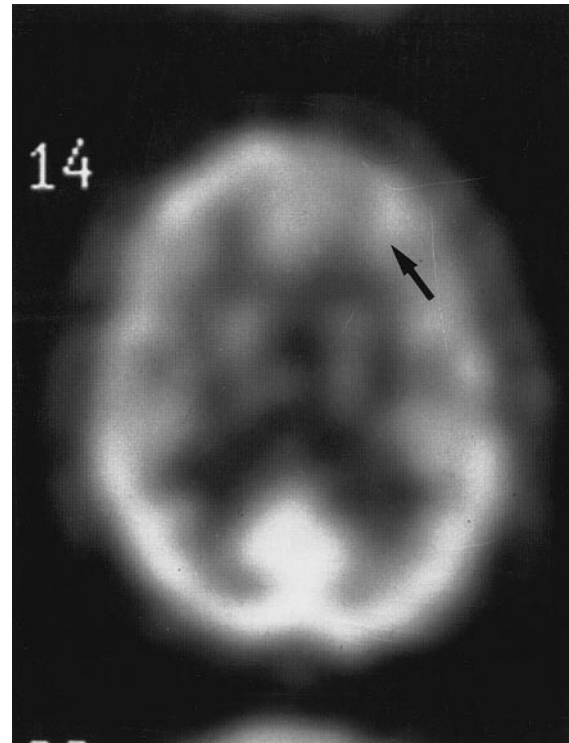


Figure 2. ECD SPECT (6 months after stroke): relative hypoperfusion of both thalami, both nuclei caudati, and mesiofrontally, most pronounced at the left (arrow).

visitors or when he received gifts. He did not show any concern for his relatives or his illness and manifested no desire, no complaint, and no concern about the future. The patient, who used to be a successful traveling salesman, had entirely lost concern about his business. There was a striking absence of thoughts and spontaneous mental activity. He rarely spoke spontaneously and took no verbal initiative. When asked about the content of his thoughts, the patient claimed he had none, suggesting a state of mental emptiness.⁴ Unless encouraged by the hospital staff or his relatives, he did not initiate any activity. When external stimulation disappeared, any induced activity was immediately interrupted. Every morning, the patient stayed in bed until he was encouraged to rise and get dressed. Once dressed, he returned to bed again or sat down in an armchair for the entire day. He moved very little unless urged to do so and reverted to his habitual state of athymia once he was left alone. There were no symptoms of depression. No stereotyped activities were observed.

A brain CT performed on the day of the acute clinical event was negative. A cerebral panarteriography was normal. A common trunk for both thalamic paramedian arteries could not be visualized. One week after admission, brain MRI showed bithalamic hyperintensities and a small hyperintensity at the right side of the upper mesencephalon. As confirmed by a neuroimaging atlas, the nucleus medialis thalami was bilaterally involved.⁸ [^{99m}Tc]hexamethylpropyleneamine-oxine (HMPAO) single-photon emission CT (SPECT) 1 week after stroke (Figure 1) disclosed luxury perfusion in both thalami and a relative

hypoperfusion of both nuclei caudati. A relative hypoperfusion of the mesiofrontal region was shown as well. These SPECT changes were most pronounced in the left hemisphere. Six months after ictus, ethyl cysteinate dimer (ECD) SPECT showed a severe relative hypoperfusion of both thalami, a relative hypoperfusion of both nuclei caudati, and a relative hypoperfusion mesiofrontally (Figure 2). Again, SPECT changes were more severe in the left hemisphere. Brain MRI 6 months after stroke showed a slightly asymmetrical bithalamic infarction with cystic transformation, more prominently involving the left thalamus (Figure 3). Mesencephalic lesions were no longer detected.

Two weeks after onset, a neurolinguistic examination revealed a transcortical-like aphasic syndrome with adynamic features. Language functions progressively improved. Six months after onset, the neurolinguistic profile disclosed only a reduced verbal fluency.

In the acute phase, the Wechsler Adult Intelligence Scale (WAIS)⁹ reflected a generalized cognitive dysfunction (global IQ of 78). Concentration, sustained attention, and frontal problem solving were impaired. A general memory disorder was objectified by means of the Wechsler Memory Scale (revised).¹⁰ The remainder of the neuropsychological examination was within normal limits.

During the next 12 months, a systematic increase in the WAIS scores showed a complete recovery of intelligence levels (global IQ of 114). Although memory, concentration, and problem-solving capacity improved, the patient did not regain his premorbid levels. Despite intensive cognitive rehabilitation, it was impossible to re-engage the

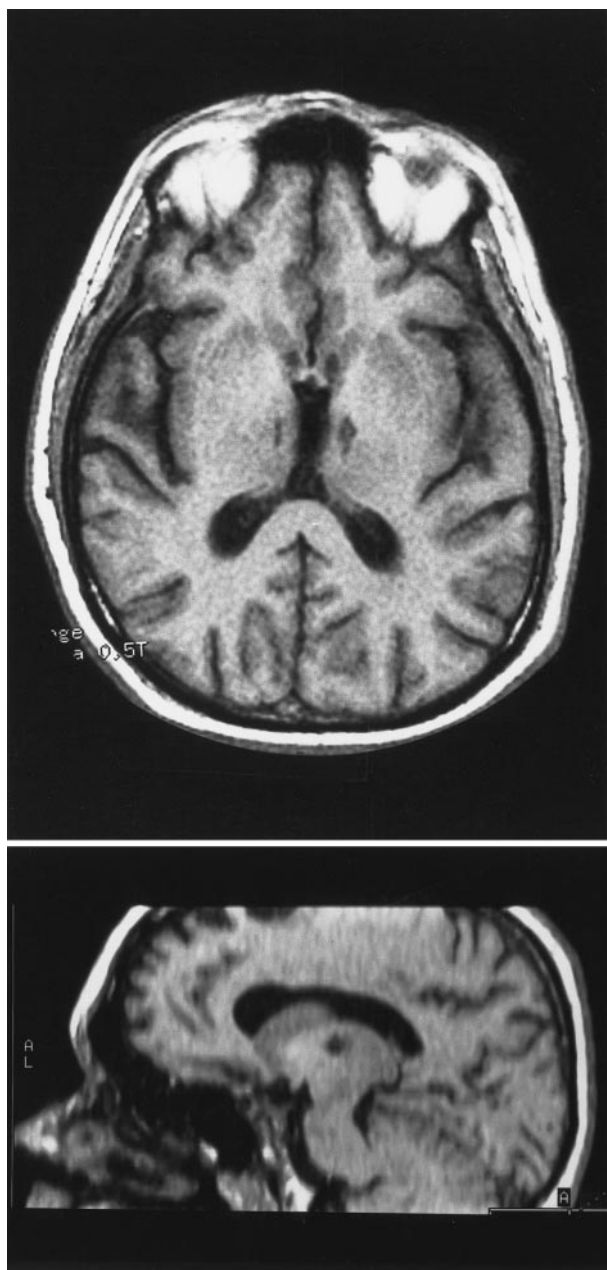


Figure 3. Brain MRI (T1-weighted images) (6 months after stroke): transverse view (top) showing an asymmetrical bithalamic infarction, more prominently involving the left thalamus; sagittal view (bottom) showing left thalamic infarction.

patient in his former professional activities because the athymic syndrome remained unchanged.

Discussion

Some of the acute-phase symptoms this patient displayed may be attributed to mesencephalic involvement. Indeed, the paramedian thalamic artery often supplies the midbrain region.¹¹ However, amelioration of these signs suggests that the mesencephalon was not part of the permanent lesion site. A follow-up MRI 6 months after the stroke did not reveal any residual mesencephalic involvement.

After recovery from the acute phase, symptomatology mainly consisted of a neurobehavioral syndrome and a

transcortical-like aphasia. Several authors have suggested that language disturbances and cognitive defects caused by thalamic lesions are due to cortical deafferentation or cortical diaschisis, which implies that functional deficits are potentially reversible.^{12–14} In our patient, the aphasic syndrome receded after the acute phase, but prefrontal adynamic speech characteristics remained.

The neurobehavioral syndrome mainly consisted of apathy, unconcern, poor motivation, and flattened affect. Because these symptoms are very similar to the clinical picture described in patients with bilateral lesions of the globus pallidus, striatum, and white matter of the frontal lobes,^{4–6} it has been suggested that bithalamic lesions can cause loss of psychic self-activation due to interruption of the striatal-ventral pallidal-thalamic-frontomesial limbic loop.⁷

SPECT and positron emission tomography studies have revealed major functional effects in frontal areas after even discrete lesions in the nucleus medialis thalami.^{15,16} It is probable that bilateral disruption at different levels of the striatal-ventral pallidal-thalamic-frontomesial limbic loop leads to a similar clinical picture consisting of loss of psychic self-activation. Our findings fully corroborate this hypothesis, because SPECT revealed a relative hypoperfusion of both basal ganglia and the bifrontal region in addition to the perfusion deficit at both thalami. Because clinical symptoms and SPECT changes persisted after a follow-up period of 18 months, it is unlikely that the neurobehavioral syndrome of our patient was caused by metabolic changes in remote brain regions as described after cerebral infarction.¹⁷ One cannot exclude the possibility that metabolic changes have determined the symptomatology and SPECT changes in 2 previously described patients, because SPECT findings were obtained respectively 16 and 20 days after stroke,⁷ and metabolic changes in remote brain regions have been reported up to 3 months after stroke.¹⁷

In conclusion, we report a case with a prominent residual loss of psychic self-activation after bilateral paramedian thalamic infarction documented during an 18-month follow-up period. SPECT findings evidenced that the neurobehavioral syndrome was most likely caused by disruption of the striatal-ventral pallidal-thalamic-frontomesial limbic loop. An 18-month follow-up based on solid clinical, neuropsychological, and neuroimaging data and the absence of additional symptoms to the loss of psychic self-activation render this case unique.

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