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Neuroimaging and cognitive changes during déjà vu

Norbert Kovacs [a,*], Tibor Auer [a,b,c,d], Istvan Balas [b], Kazmer Karadi [a,e], Katalin Zambo [f], Attila Schwarcz [b,d], Peter Klivenyi [g], Hennric Jokeit [h], Krisztina Horvath [a], Ferenc Nagy [a], Jozsef Janszky [a,d]

[a] Department of Neurology, University of Pecs, Pecs, Hungary
[b] Department of Neurosurgery, University of Pecs, Pecs, Hungary
[c] Biomedizinische NMR Forschungs GmbH am Max-Planck-Institut für Biophysikalische Chemie, Göttingen, Germany
[d] Pécs Diagnostic Institute, University of Pecs, Pecs, Hungary
[e] Institute of Behavioral Sciences, University of Pecs, Pecs, Hungary
[f] Department of Nuclear Medicine, University of Pecs, Pecs, Hungary
[g] Department of Neurology, University of Szeged, Szeged, Hungary
[h] Swiss Epilepsy Center, Zurich, Switzerland

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ABSTRACT

Objective: The cause or the physiological role of déjà vu (DV) in healthy people is unknown. The pathophysiology of DV-type epileptic aura is also unresolved. Here we describe a 22-year-old woman treated with deep brain stimulation (DBS) of the left internal globus pallidus for hemidystonia. At certain stimulation settings, DBS elicited reproducible episodes of DV.
Methods: Neuropsychological tests and single-photon-emission computed tomography (SPECT) were performed during DBS-evoked DV and during normal DBS stimulation without DV.
Results: SPECT during DBS-evoked DV revealed hyperperfusion of the right (contralateral to the electrode) hippocampus and other limbic structures. Neuropsychological examinations performed during several evoked DV episodes revealed disturbances in nonverbal memory.
Conclusion: Our results confirm the role of mesiotemporal structures in the pathogenesis of DV. We hypothesize that individual neuroanatomy and disturbances in gamma oscillations or in the dopaminergic system played a role in DBS-elicited DV in our patient.

1. Introduction

Déjà vu (DV) is "any subjectively inappropriate impression of familiarity of present experience with an undefined past" [1]. Although 60–80% of the healthy population have experienced déjà vu [2], DV aura is one of the leading symptoms of temporal lobe epilepsy (TLE) [3] occurring in 10% of all epileptic auras [4]. DV aura is the most characteristic symptom of familial mesial temporal lobe epilepsy reported in about one-third of these patients [5,6]. DV occurring in other brain disorders (e.g., depression [7] and schizophrenia [8]) has also been analyzed in more detail.

Studying DV is difficult because of its rarity, unpredictable appearance, and heterogeneity. Contrary to spontaneous DV, induced DV can be examined objectively during presurgical evaluation of epilepsy [3]. Stimulation of the temporal structures [9] or the rhinal cortex [10] often, but not always [11], elicits DV in patients with TLE. Most studies have reported that DV was confined to the nondominant temporal lobe and accompanied by hallucinations or illusions [3,4,9,11]. Furthermore, DV can also be provoked by electrical stimulation of brain structures contralateral to the epileptic focus, suggesting DV can also be elicited in normal brain tissue [12].

Despite numerous investigations, the pathomechanism of DV in healthy people remains unknown. The "small seizure" theory is based on the clinical finding that DV is an aura type in TLE. It is hypothesized that in the nonepileptic population, a "small temporal lobe seizure" may elicit DV without producing clinical seizures [13,14]. However, there are several counterarguments to this theory: DV is much more common than TLE [15,16], and only a portion of patients with TLE experience DV auras [17].

The "tape recorder" theory [18] is one of the best known DV theories applying the dual-processing approach. It assumes that

* Corresponding author. Fax: +36 72 535 911.
E-mail address: kovacsnorbert06@gmail.com (N. Kovacs).

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two different memory-related processes that normally work synchronously become asynchronous or one process becomes activated in the absence of the other. Under normal conditions, memory encoding (“recording head”) and memory retrieval (“playing head”) work with appropriate timing and synchronization. According to this speculation, if the new sensory information is simultaneously encoded and retrieved, the sensory input is accompanied by familiarity, resulting in a feeling of DV. Based on clinical evaluation of the electrically evoked DV experiences of 16 patients with TLE who underwent presurgical depth electrode implantation, Bancaud and colleagues [9] postulated the neuroanatomical bases for the tape recorder theory. Because association cortical and limbic areas encode the holistic memory of an event, and perceptual information is encoded by the temporal neocortex and stored in the hippocampus, the inappropriate activation of these centers can lead to the experience of DV. Similar electrophysiological results [19] expanded Bancaud’s theory with the complementary assumption of parallel neuronal networks underlying encoding and retrieval [20].

Interestingly, a recent case study described “drug-induced” DV, in which a patient experienced recurrent DV after receiving a combination of amantadine and phenylpropanolamine [21]. Because both drugs can facilitate dopaminergic neurotransmission and recent animal studies have proved that hippocampal dopaminergic systems are involved in spatial memory processes [22], this case suggests that increased dopaminergic activity may play a crucial role in the development of DV [21].

In a very recent case report, hypothalamic deep brain stimulation (DBS) was found to evoke detailed autobiographic memories, but not DV [23].

These data inspired us to systematically analyze the pathophysiology of DV by using functional neuroimaging (SPECT) and neuropsychological batteries in a case in which DBS of the left internal globus pallidum (GPi) elicited DV. As far as the authors are aware, this is the first study using direct, reproducible, and integrative neuropsychological and neuroimaging investigations during DV.

2. Methods

2.1. The patient

The 22-year-old female university student was born with a right-sided spastic hemiparesis due to a perinatal injury. Although the strength of the right limbs normalized, the abnormal posture of the right upper limb, observed at the age of 2 months, developed into a drug-refractory and painful secondary hemidystonia. Locomotive and intellectual development was otherwise normal.

Brain MRI revealed a $4 \times 15 \times 18$-mm lesion in the left globus pallidum. At age 22, she underwent microelectrode-guided implantation of Medtronic quadripolar 3389 DBS electrodes into the left posteroventral GPi without perioperative complications. The patient gave written informed consent to the entire surgical procedure, pre- and postsurgical examinations, and publication of this report; the study was also approved by the local ethics committee.

2.2. Stimulation settings

On the first postoperative day, contact 1 was activated in monopolar mode (C + 1–, 120 μs, 130 Hz, 3.2 V) without any adverse reactions. The patient was admitted to the neurological ward in the third postoperative week to learn how to use the patient controller. During testing of the electrodes, we noticed that monopolar stimulation of contact 0 with an amplitude exceeding 2.7 V elicited several DV episodes. Because turning on or turning off the stimulation had an immediate effect on this experience, we assumed it was a stimulation-related adverse reaction. The impedance of contact 0 (C + 0–, 3.2 V, 120 μs, 130 Hz) was 562 ohms.

2.3. Single-photon-emission computed tomography

Current safety regulations do not permit the use of functional MRI during DBS [24]. Therefore, we performed ^{99m}Tc-hexamethylpropyleneamineoxime (^{99m}Tc-HMPAO) single-photon-emission computed tomography (SPECT) to study the pathophysiology of DV because ^{99m}Tc-HMPAO binds more rapidly (2–10 minutes) compared with positron emission tomography (PET) tracers [25].

SPECT was performed 1 month postoperatively. To exclude the long-term effect of DBS, a baseline SPECT scan was obtained during normal stimulation of contact 1 (C + 1–, 3.2 V, 120 μs, 13 0 Hz). To study the pathophysiology underlying DV, 3 days later we stimulated simultaneously both contact 0 and contact 1 (C + 0–1–, 120 μs, 130 Hz, 3.2 V, referred to as *DV-inducing stimulation*). Analogously to epilepsy studies, we defined this setting as *ictal SPECT*.

As the ^{99m}TcHMPAO tracer (750 MBq) was administered immediately after starting the DV-inducing stimulation and the patient experienced numerous DV episodes during the first 5 minutes of stimulation, we assumed that the tracer binding in ictal SPECT represented the combination of acute DV induction and normal pallidal stimulation. Therefore, the subtraction of baseline from ictal SPECT images theoretically indicated those areas activated during the DV episode. Baseline and ictal SPECT images were compared using the subtraction ictal SPECT co-registered to MRI (SISCOM) method, which is also used in the presurgical evaluation of epilepsy [26].

2.4. Neuropsychological tests

The subject underwent neuropsychological examinations three times: 9 months preoperatively and 2 months postoperatively with and without DV-eliciting stimulation. There was a 1-day difference between the postoperative examinations, during which the Rey and Medical College of Georgia Complex Figure, Rey 8/64 Visual Learning, Benton Visual Retention, Boston Naming, and Rey Auditory Verbal Learning tests were administered [27,28] (see Supplementary Data).

3. Results

3.1. The occurrence of déjà vu

Preoperatively the patient had never experienced DV. Immediately after turning on the DV-inducing stimulation, she experienced an unusual and obscure feeling. In addition to discomfort and a slight disturbance, the subject had an intact sense of reality; she was able to observe what was going on around her and to maintain verbal and behavioral responsiveness. We defined this period as the *standby state for DV* (SSDV). The SSDV persisted until stimulation of contact 0 was turned off or the amplitude of stimulation was lowered below 2.7 V.

During SSDV, she experienced impulse DV episodes lasting 4–5 seconds. On these occasions she felt that the situation seemed familiar. No visual or auditory illusions or hallucinations accompanied the DV. In addition, the patient felt neither the ability to predict the future nor unreality about current circumstances.

DV occurred more frequently immediately after turning on the stimulation (approximately two to five DV episodes during the first 5–10 minutes) and became rarer as time went by (approximately another three to five DV episodes in the first hour and two or three in the second hour). Interestingly, she experienced DV only if her

eyes were open and she was questioned directly (e.g., "What is the name of your physiotherapist?"). However, not all direct questioning of the patient elicited DV. Standard digital EEG recording revealed physiological activity; there were no clinical signs of epilepsy.

3.2. Magnetic resonance imaging

Preoperative speech-activated functional MRI demonstrated right-sided language dominance (see Supplementary Data) based on a technique described previously [29]. Postoperative MRI demonstrated that the stimulating electrode passed through the GPi, which was confirmed by the fact that normal stimulation improved the severity of dystonia. Visual inspection and use of an electronic version of the stereotactic atlas [30] verified that this contact was situated between the GPi and the underlying white matter (Fig. 1). The electrode reached neither the fornix, nor the hippocampus, nor any other mesial temporal structure. The exact position of the lowest contact that could, on stimulation, elicit DV was 22 mm lateral from the midline, 13 mm anterior to the posterior commissure, and 12 mm below the intercommissural line (the distance between the anterior and posterior commissures was 24.9 mm).

3.3. Single-photon-emission computed tomography

The results of SISCOM analysis are illustrated in Fig. 2, and the hyperperfusion and hypoperfusion clusters are listed in the Supplementary Data. Compared with the baseline, SPECT during DV revealed right-sided hyperperfusion of the hippocampus, parahippocampal gyrus, fusiform gyrus, cerebellum, and temporal superior pole, and left-sided hyperperfusion of the cerebellum, operculum, insula, lingual gyrus, precuneus, and middle temporal gyrus. Hypoperfusion appeared bilaterally in the precentral and postcentral gyri, as well as in the frontal (especially supplementary motor cortex) and parietal areas.

3.4. Neuropsychological tests

The results of neuropsychological batteries are summarized in Table 1. Preoperatively the Hungarian standardized version of the Wechsler Adult Intelligence Scale (WAIS) revealed an IQ of 119 [31].

Verbal fluency, Complex Figure copy, Benton delayed memory, and Trail Making A and B scores were better during normal stimulation compared with the preoperative state. However, recall memory of the Complex Figure worsened during normal stimulation.

During DV-eliciting stimulation there was some deterioration in verbal fluency and Boston Naming Test scores. Furthermore, nonverbal memory as measured by the Complex Figure Test and Rey 8/64 Visual Learning Test was severely impaired, compared with either normal stimulation or the preoperative state.

3.5. Discussion

In our patient the electrically evoked DV phenomenon could be easily studied for several reasons: (1) it could be repeated without any restraints. (2) DV could be elicited several times. (3) No other neurological phenomenon disturbed the evaluation (e.g., altered consciousness during an epileptic seizure). (4) The anatomical site of electrical stimulation could be determined by high-resolution MRI. (5) The functional changes in the brain during DV could be identified by functional neuroimaging. (6) The examinations did not bother or harm the patient.

The main findings of our study are:

1. Pallidal DBS may evoke DV as an adverse reaction, which can be resolved by changing to a more proximal contact or reducing the stimulation amplitude.
2. The elicited DV can be characterized as "nonpathological" [15].
3. Contrary to the unreliable results of electrical stimulation in patients with epilepsy [12], in our case the setting of certain stimulation parameters reliably and reproducibly elicited DV.
4. Neuropsychological examinations indicate prominent alterations in visual learning and retrieval during DV. During normal stimulation, performance on most neuropsychological tests improved compared with the preoperative state, which may be associated with the dystonic pain-assuaging effect of normal stimulation. Conversely, DV-eliciting stimulation slightly worsened verbal memory performance and severely impaired nonverbal memory performance.
5. This is the first study in which functional neuroimaging was performed during DV.
6. SPECT analysis revealed hyperperfusion of mesiotemporal structures contralateral to the stimulating electrode during DV.

3.6. Clinical manifestation

3.6.1. Standby state for déjà vu

A surprising finding was that the pallidal DBS elicited two distinct types of symptoms: SSDV and DV. Immediately after turning on the DV-eliciting stimulation, the patient experienced a feeling of slight discomfort. This state occurred as a nonhabituating adverse reaction, which could be resolved by either changing to a more proximal contact or reducing the stimulation amplitude. Several

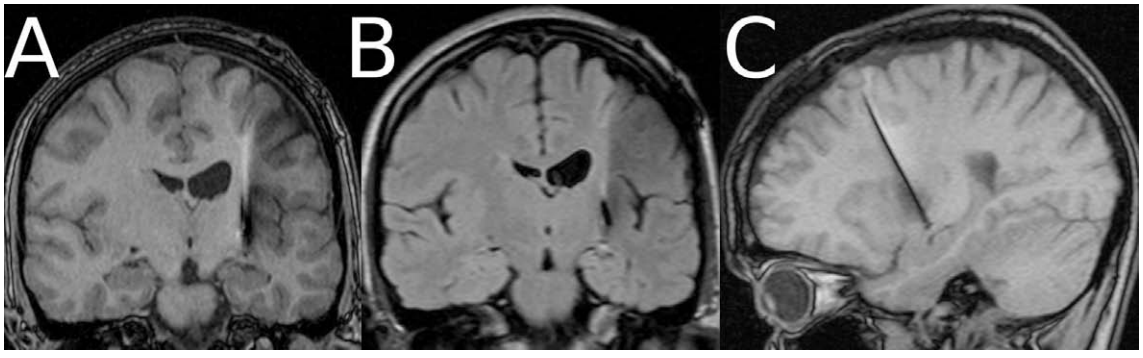

Fig. 1. Localization of the stimulating electrode: (A) coronal MP-RAGE, (B) coronal FLAIR, (C) sagittal MP-RAGE. Visual inspection and application of the electronic version of the Schaltenbrand stereotactic atlas verified that the contact responsible for DV was situated between the GPi and the underlying white matter. The electrode did not hit the mesial temporal structures.

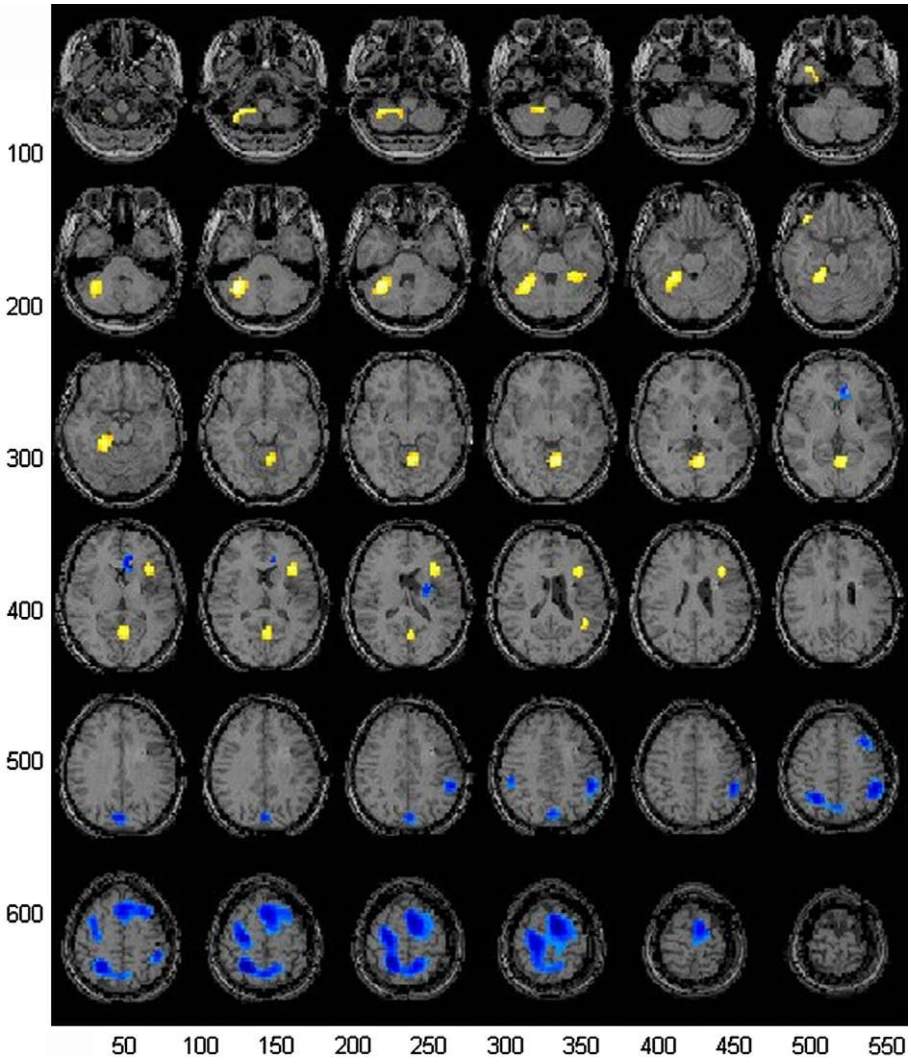

Fig. 2. SISCOM analysis comparing déjà vu-eliciting stimulation (ictal) with the normal (interictal) SPECT superimposed on axial MRI scans. Yellow: hyperperfusion, blue: hypoperfusion. Compared with baseline, SPECT during DV showed right-sided hyperperfusion of the hippocampus, parahippocampal gyrus, fusiform gyrus, cerebellum, and temporal superior pole and left-sided hyperperfusion of the cerebellum, operculum, insula, lingual gyrus, precuneus, and middle temporal gyrus. Hypoperfusion appeared bilaterally in the precentral and postcentral gyri, as well as in the frontal (especially supplementary motor cortex) and parietal areas.

Table 1
Preoperative and postoperative neuropsychological test results obtained with standard stimulation settings (C + 1–, 120 μs, 130 Hz, 3.2 V) and déjà vu-eliciting (C + 0–1–, 120 μs, 130 Hz, 3.2 V)

	Preoperative	Standard stimulation	Déjà vu stimulation
Digit Span Forward	5	4	5
Corsi Test	4	4	4
Trail Making Test (A/B)	35 s/71 s	24 s/44 s	27 s/48 s
Verbal fluency	F: 12, A: 8, S: 9, category: 26	P: 17, E: 16, M: 13, category: 17	F: 9, A: 9, S: 7, category: 20
Boston Naming Test	Not obtained	60	57[a]
Auditory–Verbal Learning Test	Learning phase: 47 recalled words; New information: 7; Interference: 10; Delayed Recall: 10; Recognition: 100%	Learning phase: 49 recalled words; New information: 4; Interference: 9; Delayed Recall: 11; Recognition: 100%	Learning phase: 54 recalled words; New information: 3[a]; Interference:12; Delayed Recall: 14; Recognition: 100%
Rey/Medical College of Georgia Complex Figure (copy/recall)	30/26	35/18	36/12
Benton Visual Retention Test	C-figure memory: 6/10; D-figure copy: 10/10	E-figure delayed recall: 8/10	C-figure: 7/10; D-figure delayed recall: 6/10[a]
Rey 8/64 Visual Learning Test	Not obtained	Learned by sixth trial	No learning

[a] While the subject was performing these tests, she experienced a sudden feeling of déjà vu lasting several seconds.

times we turned on or off the stimulation of contact 0 and decreased or increased the stimulation voltage across the threshold level, but the patient always indicated the presence or absence of SSDV without exception.

Although normal stimulation improved overall neuropsychological function as compared with the preoperative state, DV-eliciting stimulation worsened cognitive (mainly memory) function. Because the dystonic pain had been eased equally by the time of the postoperative neuropsychological tests, the neuropsychological differences between normal and DV-eliciting stimulation were probably due to the different stimulation settings and were unrelated to the impact of pain on attention. Interestingly, these disturbances during SSDV did not interfere significantly with everyday functioning; the subject had an intact sense of reality and maintained verbal and behavioral responsiveness.

3.6.2. Déjà vu

The actual DV episode occurred suddenly without a prodrome and lasted 3–5 seconds during which the patient could talk. The fact that open eyes and direct questioning were required to elicit DV indicated that a certain level of arousal and/or visual stimuli is needed for DV.

The occurrence of DV seemed to have a habituating feature; that is, DV occurred more frequently during the first 5 minutes of stimulation (two to five episodes) of contact 0 and became rarer with time.

Because DV is a transitory experience lasting a few seconds, we could not administer neuropsychological tests targeting it directly. However, on the "ictal" SPECT scan we could identify the brain structures involved in DV, as the patient experienced several DV episodes during the interval of tracer binding. Therefore, subtraction of ictal from baseline images presumably indicates those areas responsible for DV.

3.7. Neuroanatomical considerations

Based on the position of the stimulation electrode, we might speculate on the anatomical target responsible for DV. Postoperative MRI scans demonstrated that contact 0 was situated on the border between GPi and the underlying white matter. The spread of electrical current is roughly spherical around the activated contact and in no case extends underneath the electrode [32]. We can also presume that the electricity can diffuse approximately up to 4 mm in a low-impedance tissue from the surface of the contact [33]. Because mesial temporal structures (e.g., hippocampus and fornix) are situated bellow the lowest contact, direct stimulation of these mesiotemporal structures was unlikely.

We performed subtraction SPECT analysis comparing ictal and baseline SPECT images. The resulting picture revealed hyperperfusion of the right mesial structures contralateral to the stimulation, as well as ipsilateral (left) operculum, insula, precuneus, and lingual gyrus. This finding is in accord with TLE studies [9] demonstrating that elicitation of DV involves mainly mesiotemporal structures.

3.8. Proposed theories on the pathophysiology of DV

We cannot explain the pathophysiology of DBS-evoked DV. However, we can provide some possible theories on the basis of our results.

As far as the authors are aware, there is not even a single published report describing the occurrence of DV after pallidotomy, even though tens of thousands have been performed worldwide [34,35]. Because ablative procedures could not evoke DV and certain stimulation settings were required to produce DV, we may presume the importance of high-frequency stimulation in the background.

1. We can hypothesize that several independent constellations together led to DV: (i) the altered memory functions of the SSDV and a certain combination of (ii) visual and (iii) direct verbal stimuli requiring memory matching processes. This hypothesis is based on the fact that the DV-inducing stimulation itself was unable to produce DV in the absence of simultaneous visual and verbal stimuli; it elicited "only" the SSDV with nonverbal memory disturbances. Clinically, DV occurred only when the patient was addressed with questions and her eyes were open. The type of visual stimuli seemed to be irrelevant in the elicitation, because DV occurred in both dim and bright rooms, with or without persons in the visual field. On the contrary, only direct questioning of the patient was able to elicit DV; other auditory stimuli (e.g., environmental noises, conversation between other persons not involving the patient) never did so. However, not all questioning elicited DV. One potential explanation for this phenomenon might be that direct questioning requires simultaneous high-level attention, interpretation, and memory processing.

2. Probably not only left GPi stimulation itself, but also individual (atypical) neuroanatomy might play a role in the development of DV. The atypical language dominance suggests that because of her perinatal brain injury, our patient developed an atypical brain anatomy [36–39]. Because reorganization of the injured brain is not always complete, similar modalities might be present bilaterally and the division of labor between dominant and nondominant hemisphere functions might not be complete. Normally, the language-dominant hemisphere is more strongly engaged in memory processing of verbal material [40]. However, in our case the neuroanatomy may be such that nonverbal functions are confined to both hemispheres. If this speculation is true, the disturbing effect of DBS of the left GPi may have greater impact on one hemisphere and less on the other, producing DV as described in dual-processing theories. The elicitation of DV seemed to be a habituating phenomenon. An explanation for this habituation may be that the memory processing system(s) recognized the DV episodes as errors. Possibly, this error recognition enabled the system to adapt to the SSDV, resulting in fewer DV episodes over time.

3. The dopaminergic system may also play a role in the elicitation of DV. As discussed earlier, the concomitant use of amantadine and phenylpropanolamine was reported to have produced recurring DV [21]. On the basis of PET studies carried out on patients with Parkinson's disease, it is believed that pallidal DBS might interfere with endogenous dopamine release and/or dopamine receptor functions [41]. A recent study investigating drug effects on schizophrenic patients found that the dosage of antipsychotic (antidopaminergic) drugs is positively correlated with the frequency of DV episodes [42], which contradicts the theory that DV is a result of elevated dopaminergic activity, but underlines the role of the dopaminergic system in the pathophysiology of DV. Therefore, we cannot rule out the possibility that it was not the stimulation itself, but the disturbances in the dopaminergic system that were responsible for DV. However, one could expect that considerably more time would be needed to alter the dopaminergic system.

4. Another hypothesis might be that DV is caused by separation of two main memory systems: familiarity and recollection. Recent neuropsychological data suggest that recognition memory operates by two different mechanisms: recollection and familiarity discrimination [43,44]. Therefore, in certain situations it is possible to recognize that a person or a subject is familiar even without the ability to recollect any particulars about it. Electrophysiological studies in monkeys have demonstrated that neurons in the rhinal cortex respond differently to familiar and novel stimuli, and this occurs more rarely in the hippocampus [45]. Therefore, possibly two separate memory compartments may coexist: one, including the hippocampus, enables recall and conscious recollection of contextual elements, whereas the other system, including the structures of the parahippocampal gyrus, is important for familiarity judgments [46,47]. In our case, the DV-eliciting stimulation altered

the nonverbal memory processes, including recollection, as assessed by neuropsychological tests. Thus, during DV both recollection and familiarity discrimination were affected, which contradicts the concept that DV is the result of a separation of these two memory systems. We might presume that the elicitation of DV is the result of the altered dual processing caused by atypical functional localization of recollection and familiarity systems. Possibly, the heavy memory-related load induced by the visual and auditory stimuli produces a delay in memory processing between the hemispheres, resulting in false familiarity recognition.

5. The nondominant localization of the GPi-stimulating electrode may also have had an impact on elicitation of DV. Previous electrophysiological studies confined the DV to the nondominant hemisphere; however, these studies were limited to TLE [9,19].

6. Furthermore, experimental studies have demonstrated a functional relationship between the hippocampus and the contralateral basal ganglia [48]. In rats, electrical stimulation of globus pallidum alters contralateral hippocampal theta field activity presumably via a septohippocampal pathway. This relationship between the GPi and the hippocampal formation has not been verified in humans yet. However, there are some indirect data supporting this hypothesis. For example, the transitory unilateral ictal dystonia in TLE could be associated with hyperperfusion of the basal ganglia ipsilateral to the seizure focus [49]. Alternatively, GPi stimulation could be accompanied by hypoperfusion of the mesiotemporal structures [50].

7. Analogously we can assume that high-frequency DBS can interfere with high-frequency (gamma) oscillations of the contralateral mesiotemporal structures, which, in turn, play a crucial role in memory functions. Phase synchronization of gamma oscillations of around 40–50 Hz is a general mechanism underlying transient functional coupling between different neuroanatomical structures playing an important role in every aspect of memory functions [51–53]. Thus, one may expect that high-frequency DBS can interfere with gamma oscillations in the brain independent of the stimulating site. Moreover, novel studies hypothesize that DV may be related to the alteration of gamma oscillations of mesiotemporal structures [51].

8. Because the major output from GPi is to thalamus and from there to cortex, its role in the development of déjà vu should also be considered. However, several thousands of GPi DBS electrodes have been implanted worldwide either for Parkinson's disease or for dystonia, and not even a single case report has mentioned DV as a stimulation-related side effect. Therefore, the sole alteration of pallido-thalamico-cortical pathways is unlikely to produce DV.

In a recent case report, Hamani et al. reported that hypothalamic DBS evoked detailed autobiographic memories [23]. The stimulation increased recollection, but not familiarity-based recognition, nor DV. EEG source localization suggested activity in the mesiotemporal structures [23]. Considering our patient together with Hamani and colleagues' patient, we may assume that in the case of certain constellations (e.g., specific electrode localizations, stimulation parameters, and individual neuroanatomy), deep brain stimulation can interfere with some memory-related processes. However, these types of memory alterations due to deep brain stimulation are rather rare.

3.9. Open questions and limitations

Several questions remain unanswered in our case. We compared only the functional neuroimages of DV-eliciting and "normal" pallidal stimulation. Because during tracer binding four DV episodes occurred, the differences between these scans probably identify the anatomical structures responsible for DV. However, we should have obtained a third SPECT scan during SSDV without DV during tracer binding to compare activation between the DV and SSDV and between the SSDV and normal stimulation. In this way, we could have identified those structures that are directly responsible for the DV experience but do not contribute to the SSDV. Moreover, the SPECT scan methodology (SISCOM) was adapted from epilepsy studies, and is usually useful for seizures lasting more than 20–30 seconds. The single observation in this patient of a DV episode lasting less than 10 seconds is of uncertain significance. To test reproducibility, we had planned to repeat baseline (normal stimulation) and ictal (DV-eliciting stimulation) SPECT, but in respecting the request of our patient, we decided not to repeat the scans.

Conflict of interest statement

None of the authors has a conflict of interest to disclose.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.yebeh.2008.08.017.

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