Contents lists available at ScienceDirect

Epilepsy & Behavior Case Reports

journal homepage: www.elsevier.com/locate/ebcr

Case Report

Déjà vu phenomenon-related EEG pattern. Case report ☆

P.N. Vlasov [a], A.V. Chervyakov [b,*], V.V. Gnezditskii [b]

[a] Moscow State University of Medicine and Dentistry, Moscow, Russia
[b] Research Center of Neurology, Russian Academy of Medical Sciences, Moscow, Russia

ABSTRACT

Background: Déjà vu (DV, from French déjà vu — "already seen") is an aberration of psychic activity associated with transitory erroneous perception of novel circumstances, objects, or people as already known.
Objective: This study aimed to record the EEG pattern of déjà vu.
Methods: The subjects participated in a survey concerning déjà vu characteristics and underwent ambulatory EEG monitoring (12–16 h).
Results: In patients with epilepsy, DV episodes began with polyspike activity in the right temporal lobe region and, in some cases, ended with slow-wave theta–delta activity over the right hemisphere. There were no epileptic discharges in healthy respondents during DV.
Conclusion: Two types of déjà vu are suggested to exist: "pathological-epileptic" déjà vu, characteristic of patients with epilepsy and equivalent to an epileptic seizure, and "nonpathological-nonepileptic" déjà vu, which is characteristic of healthy people and psychological phenomenon.

© 2013 The Authors. Published by Elsevier Inc. All rights reserved.

1. Introduction

Déjà vu (DV, from French déjà vu — already seen) is the term describing an aberration of psychic activity associated with transitory erroneous perception of novel circumstances, objects, or people as already known. This phenomenon belongs to the group of derealization disorders, which also includes states such as déjà vécu (already experienced), déjà entendu (already heard), and jamais vu (never seen). According to another classification, DV is a memory-based illusion [1]. The term was coined by the French psychologist Emile Boirac (1851–1917) in his monograph L'Avenir des sciences psychiques (The Future of Psychology, 1918).

The DV phenomenon attracts special interest because, on the one hand, it occurs in most healthy individuals (up to 97% of the general population), spontaneously or in association with sleeping disorders or anxiety. On the other hand, it can be a sign of certain psychoneurological diseases, such as Charles Bonnet syndrome, temporal lobe epilepsy (TLE), depression, or schizophrenia, and it can be an early symptom of a mass lesion of the brain [2–5]. Thus, DV is observed both in healthy people and in patients with organic brain damage or dysfunction. Under these circumstances, it seems reasonable to identify differential diagnostic criteria to discriminate whether DV represents a normal phenomenon or a sign of a disease.

A number of modern publications are concerned with the mechanisms underlying DV and its characteristics and prevalence [2,4–8]. We also described major clinical differential diagnostic characteristics of DV in healthy individuals [9], in patients with mass lesions of the brain [10], and in patients with epilepsy [9].

Déjà vu is particularly interesting as a sign of epilepsy. A DV aura occurs in 10% of patients with TLE [11]; DV as a sign is present in 2/3 of patients with idiopathic generalized epilepsy [12]. In our previous paper, we also describe DV in patients with idiopathic generalized epilepsy [13].

Autosomal dominant temporal lobe epilepsy (ADTLE) is characterized by focal seizures with auditory symptoms or aphasia. More than 50% of patients with ADTLE have an LGI1 mutation. Recently, ADTLE cases with psychic presentations (DV and fear) but lacking classic aphasia and auditory symptoms have been described. These patients had a previously unknown LGI1 mutation, Arg407Cys, which, in contrast to the mutations described earlier, did not prevent protein secretion in vitro [14]. To identify the brain regions involved in DV, patients with TLE with and without DV episodes were investigated using voxel-based analysis of ^{18}FDG-PET brain scans. Patients with TLE with DV episodes exhibited unilateral focal hypometabolism in the superior temporal gyrus and the parahippocampal region, in the vicinity of the perirhinal and entorhinal cortices [15].

Gloor [16] implanted electrodes on 35 patients with TLE with pharmacoresistant seizures and found that most DV episodes were associated with stimulation of the right hemisphere. Ide et al. [17] performed a SPECT investigation in a patient with frequent DV auras

* Corresponding author at: Marshala Katukova street, 13-2-124, 123181 Moscow, Russia.
E-mail address: tchervyakovav@gmail.com (A.V. Chervyakov).

http://dx.doi.org/10.1016/j.ebcr.2013.08.001

and detected hyperperfusion in the right temporal and frontal lobes. Following pharmacotherapy, the frequency of seizures and DV episodes decreased, and the perfusion characteristics returned back to normal.

The main clinical problem unsolved is whether DV can be considered as a sign of epilepsy. Neppe [18] found that DV occurred more frequently in patients with TLE (86%) than in control individuals (68%) but did not show the significance of this difference. Some other authors argue that DV can occur as a simple partial seizure, as a part of a complex partial seizure, or as an aura of a secondarily generalized tonic–clonic seizure [19].

Apparently, the problem cannot be solved without describing the specific ictal EEG pattern of DV, which has not been done so far to our knowledge.

The aim of the present study was to investigate the electroencephalographic characteristics of the DV phenomenon in patients with epilepsy and in healthy subjects.

2. Materials and methods

We performed EEG monitoring in 20 healthy volunteers with frequent DVs and in 23 patients with epilepsy. In the course of ambulatory EEG monitoring, DV episodes were registered in one healthy volunteer and in three patients with epilepsy. We did not find any description of EEG patterns of DV in the available literature.

2.1. Ambulatory EEG monitoring (Holter EEG)

This investigation involves autonomous registration of EEG on a memory card of the recording device, without phono- or photostimulation, while the patient is free in his movement and activities, and independent of the computer. This investigation was performed in all patients with epilepsy and in 20 healthy volunteers. It provides a possibility to detect the pattern of rare seizures (including DV) during a subject's normal wakefulness. We used the 10/20 electrode placement arrangement.

3. Results

3.1. Observation examples

3.1.1. Healthy subject S., 20 years, female
For a long time (since the age of 15), the subject had been complaining of moderate tension headaches with a predisposition to meteosensitivity. At approximately the same age, she began to experience DV episodes, which had been growing more frequent.

At examination, the patient complained of headaches. Déjà vu episodes occurred several times per day, lasting up to 10 s, and were accompanied by positive emotions (surprise, interest) and no fear. Neurological examination showed signs of vegetative parasympathetic dysfunction, in particular, acrohyperhidrosis. Brain MRI did not show any pathology. Doppler ultrasound of main head arteries was according to the patient's age. A routine EEG showed moderate diffuse changes of bioelectric brain activity, without epileptiform signs. A 24-hour ambulatory EEG monitoring registered a DV episode.

At 00:42, the patient was in the kitchen, when she experienced a feeling of unreality and expected a DV. She pressed the marker button. She felt interest and pleasant emotions and became attentive to her condition. She had a feeling that everything had happened before and knew what was going to happen next (anticipation). The episode lasted about 10 to 15 s (Fig. 1).

As visible in the EEG fragment, there were no epileptiform changes during this DV episode in a subject without epilepsy. The observation showed rhythm desynchronization. The data suggest that DV in healthy subjects is basically a nonepileptic phenomenon.

3.1.2. Patient D., 29 years, male
The childhood history of patient D. was normal. Since the age of 15, D. has been experiencing rare DV episodes, such as perceiving things as

already seen in the same circumstances, up to 5 s long. At the age of 24, he suffered a closed craniocerebral injury and cerebral concussion as a result of a traffic accident. Six to seven months after the injury, the patient began to suffer from generalized tonic–clonic seizures with tongue bites, recurring about five times per year. Déjà vu episodes became more frequent and more vivid. In April 2011, D. was evaluated at the Research Center of Neurology. At that time, seizure frequency had increased to once per month. Déjà vu episodes occurred once or twice per week, lasting up to 30 s, and were accompanied by fear and unpleasant feelings; however, the patient was willing to relive the experience. A neuroimaging study did not detect any pathology. A routine EEG showed rhythm disorganization with moderately decreased amplitude and paroxysmal activity, predominantly frontal and central, more on the left, enhanced by hyperventilation.

During a 12-hour EEG monitoring, at 23:30, the patient experienced derealization and anxiety, followed by DV accompanied by an unpleasant feeling of anguish. The episode lasted up to 32 s.

The episode was accompanied by the following changes in EEG (Figs. 2, 3, 4).

Several fractions of a second before the patient pressed the button (the beginning of the seizure), a galvanic skin response was registered. The dominant alpha rhythm changes to a polymorph epileptiform activity with slow waves of theta band and sharp waves (Fig. 2).

The amplitude of the slow and sharp waves gradually increased up to 200 mV as the episode began. Epileptiform activity predominated in the right temporal lobe. The recruitment phenomenon was registered (Fig. 3). Fig. 4 shows a perspective view of the patient's seizure. The total duration of the seizure was 32 s. The pathological activity lasted for a total of 32 s, after which it was replaced by background activity.

A dipole localization procedure using the BrainLock program localized the focus of both the initial activity and the subsequent slow-wave activity in the medial temporal lobe and the medial frontal lobe of the right hemisphere (Fig. 5). In two other ambulatory EEG records of DV episodes, the polyspike activity lasted for 8 s and also showed distinct right hemisphere lateralization, predominantly in the temporal lobe.

4. Discussion

The major questions addressed in DV investigations are its clinical significance (e.g., whether it is a pathological phenomenon) and, accordingly, the necessity of treatment and the mechanisms of its generation.

The clinical and EEG characteristics of DV were fully described in our previous paper [13].

Naturally, particular attention is drawn to DV episodes in healthy individuals. Most authors believe that DV is a neurologic analog of a seizure involving psychoactive zones, rather than a psychopathological phenomenon [1].

An original hypothesis of DV generation was proposed by Spatt [8]. It assumes that the functions of the hippocampus and the prefrontal cortex involve recognition of new information and relating it to previous experience. The parahippocampal system coordinates the comparison, and aberrations of its functioning cause novel information to seem familiar, i.e., produce a DV. It was concluded that DVs occur as a result of an impaired contact between the neocortex and the medial temporal lobe structures, when the cortical influence weakens (e.g., during sleep, fatigue).

The DV states described in our study were heterogeneous by origin. Electroencephalography (EEG) results together with clinical, psychological, and neuroimaging data, suggest that two DV types exist: pathological and nonpathological.

On the other hand, Brázdil et al. [20] show that there is a difference in brain structure between subjects who have felt DV and those who have never felt DV. Authors investigated differences in brain morphology between healthy subjects with and without DV using source-based morphometry, a novel multivariate neuroimaging technique. The analysis
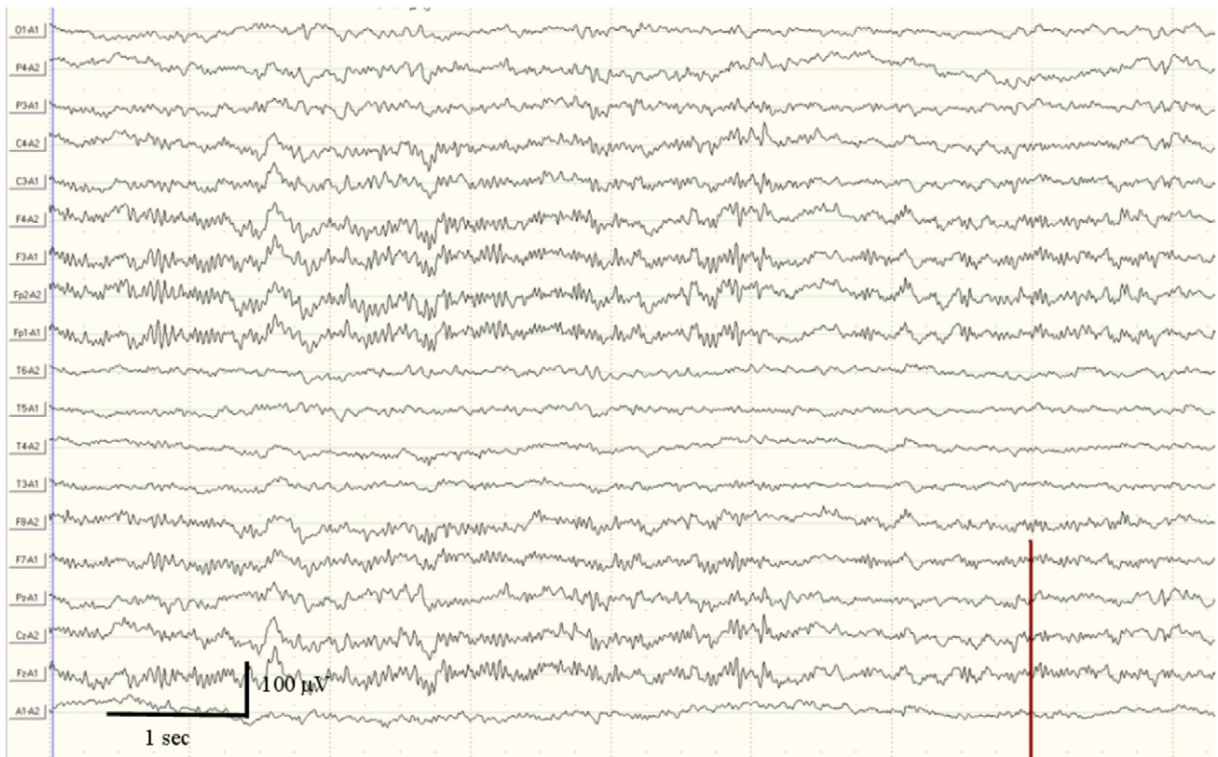
Fig. 1. EEG monitoring of patient S. A DV episode. Patient's label (red line). EEG shows rhythm desynchronization but no epileptiform signs.

revealed a set of cortical (predominantly mesiotemporal) and subcortical regions in which there was significantly less gray matter in subjects reporting DV. In these regions, gray matter volume was inversely correlated with the frequency of DV [20].

Most healthy individuals have the nonpathological-nonepileptic DV type, which does not show an epileptiform EEG pattern, has low frequency and low duration, and occurs mostly as an induced phenomenon. However, it cannot be ruled out completely that such
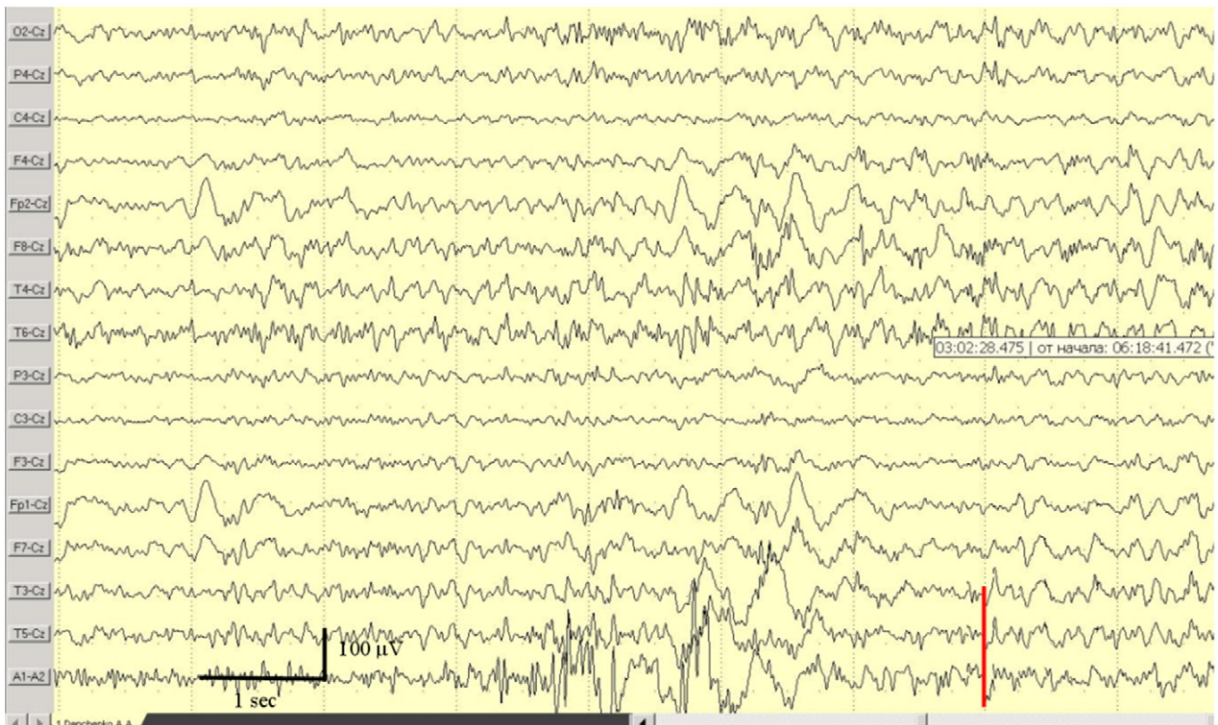

Fig. 2. Patient D., 29 years. The beginning of a DV episode. The red line is the patient's label (10 μV; 30 mm/s).
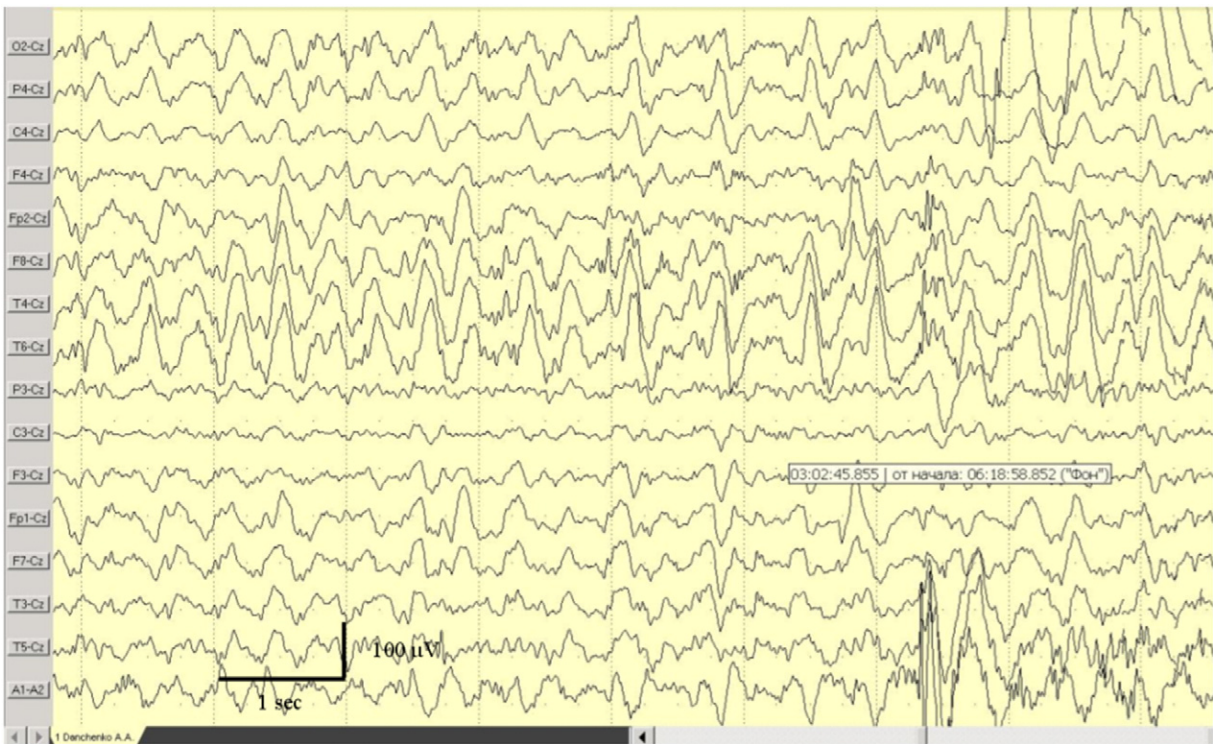
Fig. 3. Patient D., 29 years. A DV episode, continuation (10 μV; 30 mm/s).

nonpathological DV might be associated with aberrations of neuron functioning and spontaneous neuron discharging. However, the activity is probably restricted to such a small area (within the parahippocampal zone) that it does not produce any distinctive EEG pattern. On the other hand, pathologic epileptic DVs occurring in epilepsy are characterized by a specific pattern of EEG activity and typical clinical features, such as increased frequency and duration and negative emotional perception. That is, a pathological DV occurs as a result of excessive mass neuron discharges and is, in fact, a simple partial psychogenic seizure.
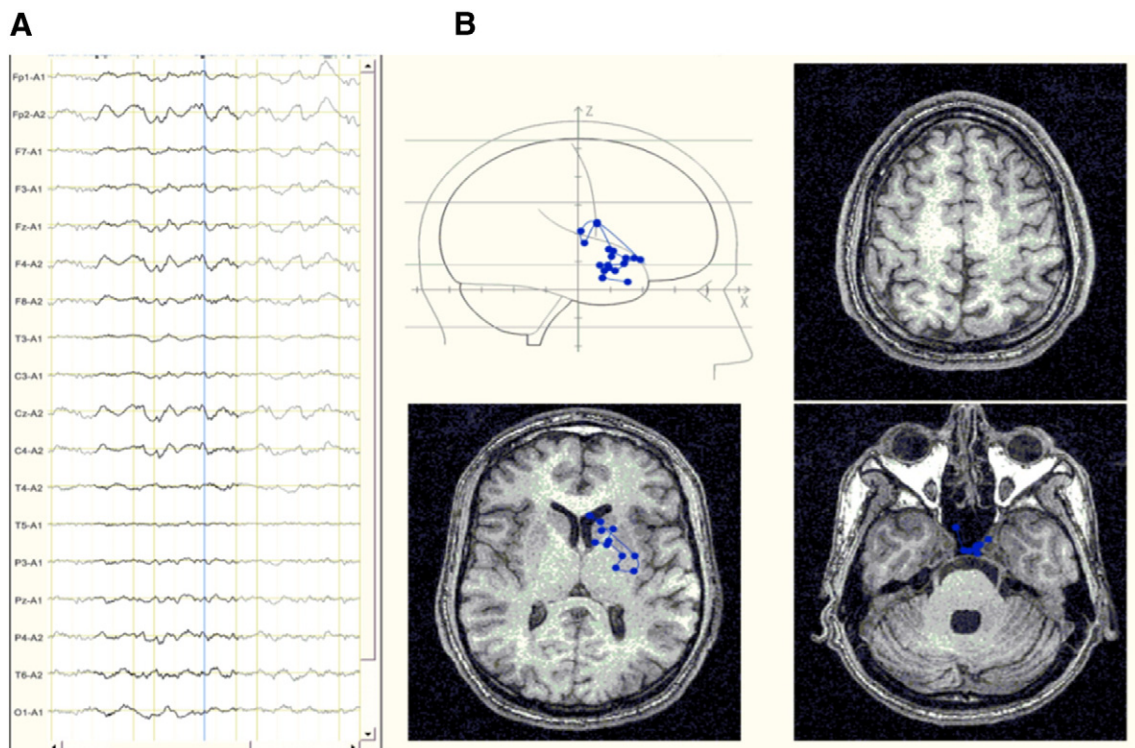

Fig. 4. Patient D., 29 years. A DV episode, general view (10 μV; 5 mm/s).
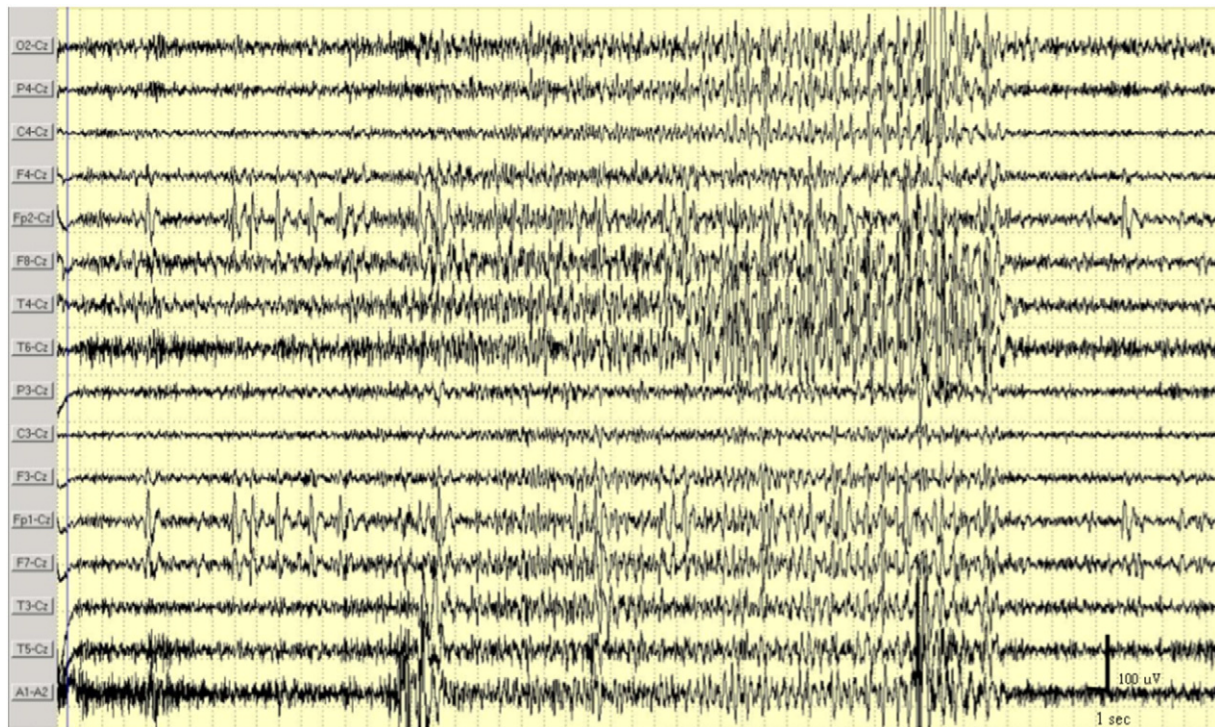
Fig. 5. BrainLock-assisted dipole-localization of a pathologic slow-wave activity focus during a DV episode in patient D., 29 years. A — an EEG fragment; B — dipole localization with the BrainLock program. Blue dots indicate the location of activity superposed on standard template MRT scans. Distinct right-hemisphere lateralization with predominating activity in the medial right temporal lobe.

Other authors also proposed a similar division with two DV types, although they did not provide any electrophysiological evidence to support the notion [10]. Our case reports confirmed our colleagues' theory.

The EEG data suggest that DV generation largely involves the right hemisphere. However, two EEG records of DV episodes showed polyspike activity in the right hemisphere, lasting for 8 s, while in a longer episode, polyspike activity was followed by slow-wave activity. It is possible that DV is not generated in any single hemisphere but results from impairment in their interaction. Importantly, PET and SPECT studies detected hypometabolism areas in temporal lobe structures (entorhinal and perirhinal cortices) [21–23]. The slow-wave activity detected in a patient with epilepsy during a DV episode can be an electrophysiological reflection of the previously described hypoperfusion. However, among the cases analyzed, DV episodes were present not only in focal forms but also in idiopathic generalized and undifferentiated epilepsy, which can probably be explained as follows:

– Each seizure changes the functioning of individual neurons and neuronal networks (up to neuronal death). Secondary epileptogenesis is characterized by selective loss of specific GABAergic interneurons and formation of new excitatory glutamatergic pathways, which also determine future seizures [24]. Such newly formed synapses have a decreased activation threshold [25].
– In TLE, neurons most commonly affected are those of the CA1 and CA3 zones, as well as of the dentate gyrus; however, extrahippocampal zones, such as the piriform and entorhinal cortices and the amygdala [24], that is, zones, responsible for DV generation, are also involved. Thus, the patterns of neuronal damage and death and synaptic reorganization constitute the fundamental mechanism of epileptogenesis, both in animal models and in human patients with TLE [25]. Postseizure reorganization of neural networks in DV-generating

zones (irrespective of the epilepsy form) can produce the phenomenon in the absence of a particular pathologic process in this area.

Also, DV in patients with idiopathic generalized epilepsy is characterized by a frequency of 1–2 times a month and duration of 5–10 s, accompanied by positive emotions and lack of fear. So, it was similar to the DV in healthy respondents but a bit more frequent.

5. Conclusion

In patients with epilepsy, DV is equally frequent in cryptogenic and symptomatic focal epilepsy; it can accompany nearly all seizure types and occur as a simple partial seizure or as a part of a secondarily generalized seizure. Major clinical features distinguishing DV in patients with epilepsy from that in healthy individuals are its frequency, the fear preceding DV, and the emotional perception. A very important criterion is DV dynamics, such as increasing frequency and duration, or appearance of negative emotions. On EEG, DV began with polymorphic spike and slow-wave activity in the right temporal lobe. Our combined clinical and electrophysiological investigation identified two separate DV types: epileptic déjà vu, which is characteristic of patients with epilepsy and equivalent to an epileptic seizure, and nonepileptic déjà vu, which occurs in healthy individuals and is basically a psychological phenomenon.

References

[1] Illman A Nathan, Butler R Chris, Souchay Celine, Moulin JA Chris. Déjà experiences in temporal lobe epilepsy. Epilepsy Res Treat 2012:539–67.
[2] Brown AS. A review of the déjà vu experience. Psychol Bull 2003;129:394–413.
[3] Karlov VA. The modern concept of epilepsy. Journal nevrologii I psichiatrii im S.S. Korsokova, 5; 1999. p. C 4–7 [T. 99, Article in Russian].
[4] Warren-Gash C, Zeman A. Short report: is there anything distinctive about epileptic deja vu? J Neurol Neurosurg Psychiatry Jan 11 2013, http://dx.doi.org/10.1136/jnnp-2012-303520.

[5] Dobrokhotova TA, Urakov SV, Chebysheva TS. Mental disorders of tumors of the cerebral hemispheres. In the book Neuropsychiatry. Moscow: Publishing House "Bean"; 2006. p. 107–31 [Article in Russian].

[6] Adachi N, Koutroumanidis M, Elwes RDC, et al. Interictal [18]FDG PET findings in temporal lobe epilepsy with déjà vu. J Neuropsychiatry Clin Neurosci 1999;11:380–6.

[7] Bancaud J, Brunet-Bourgin F, Chanvel P, Hargren E. Anatomical origin of déjà vu and vivid "memories" in human temporal lobe epilepsy. Brain 1994;117:71–91.

[8] Spatt JMD. Déjà vu: possible parahippocampal mechanisms. J Neuropsychiatry Clin Neurosci February 2002;14:6–10.

[9] Vlasov PN, Chervyakov AV. The value of déjà vu phenomenon in healthy people. Neurol neuropsychiatry psychosom 2009;2:53–7 [Article in Russian].

[10] Vlasov PN, Chervyakov AV, Urakov SV, Lukshina AA. Diagnostic significance of déjà vu phenomenon at the clinic of brain tumors. Ann Clin Exp Neurol 2011;5(3):26–31 [Article in Russian].

[11] Palmini A, Gloor P. The localizing value of auras in partial seizures: a prospective and retrospective study. Neurology 1992;42:801–8.

[12] Adachi N, Akanuma N, Ito M, Adachi T, Takekawa Y, Adachi Y, et al. Two forms of déjà vu experiences in patients with epilepsy. Epilepsy Behav Jul 2010;18(3):218–22.

[13] Chervyakov AV, Gnezditskii VV, Vlasov PN, Kalmykova GV. EEG characteristics of déjà vu phenomenon. J Epileptol 2013;21(1):35–43.

[14] Striano P, Gambardella A, Coppola A, et al. Familial mesial temporal lobe epilepsy (FMTLE): a clinical and genetic study of 15 Italian families. J Neurol 2008;255:16–23.

[15] Guedja E, Aubert Sandrine, McGonigal Aileen, Mundler Olivier, Bartolomei Fabrice. Déjà-vu in temporal lobe epilepsy: metabolic pattern of cortical involvement in patients with normal brain MRI. Neuropsychologia 2010;4.

[16] Gloor P. Experiential phenomena of temporal lobe epilepsy. Facts and hypotheses Brain 1990;113(6):1673–94.

[17] Ide M, Mizukami K, Suzuki T, Shirashi H. A case of temporal lobe epilepsy with improvement of clinical symptoms and single photon emission computed tomography findings after treatment with clonazepam. Psychiatry Clin Neurosci 2000;54:595–7.

[18] Neppe VM. The concept of déjà vu. Parapsychol J S Afr 1983;4:1–10.

[19] Van Paesschen W, King MD, Duncan JS, Connelly A. The amygdala and temporal lobe simple partial seizures: a prospective and quantitative MRI study. Epilepsia 2001;42:857–62.

[20] Brázdil M, Mareček R, Urbáneka T, Kašpáreka T, Mikla M, Rektora I, et al. Unveiling the mystery of déjà vu: the structural anatomy of déjà vu. Cortex October 2012;48(9):1240–3.

[21] Bartolomei F, Barbeau E, Gavaret M, Guye M, McGonigal A, Régis J, et al. Cortical stimulation study of the role of rhinal cortex in déjà vu and reminiscence of memories. Neurology 2004;63(14):858–64.

[22] Engel Jr J. The timing of surgical intervention for mesial temporal lobe epilepsy: a plan for a randomized clinical trial. Arch Neurol 1999;56:1338–41.

[23] Takeda Y, Kurita T, Sakurai K, Shiga T, Tamaki N, Koyama T. Persistent déjà vu associated with hyperperfusion in the entorhinal cortex. Epilepsy Behav Jun 2011;21(2):196–9.

[24] Ben-Ari Y, Dudek FE. Primary and secondary mechanisms of epileptogenesis in the temporal lobe: there is a before and an after. Epilepsy Curr 2010;10(5):118–25.

[25] Ben-Ari Y, Crepel V, Represa A. Seizures beget seizures in temporal lobe epilepsies: the boomerang effects of newly formed aberrant kainatergic synapses. Epilepsy Curr 2008;8:68–72.