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Electroencephalographic Characteristics of the Déjà Vu Phenomenon

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Déjà vu (DV) – is an aberration of mental activity associated with the perception of surrounding reality with the impression that unknown objects, new contexts, and people seen for the first time are for some moments perceived as familiar. The aim of the present work was to study the EEG characteristics of the DV phenomenon in epilepsy. A total of 166 subjects took part in the study; subjects were 25.17 ± 9.19 years old and 63.2% were women. The déjà vu phenomenon was compared in groups of healthy subjects (139 subjects) and epilepsy patients (27 cases). Patients were interviewed regarding the characteristics of DV and underwent prolonged (12–16 h) ambulatory EEG monitoring. On the EEG, the phenomenon of DV was characterized by onset with multispike activity in the right temporal leads and, in some cases, ended with slow-wave θ–δ activity in the right hemisphere.

Keywords: déjà vu, epilepsy, all-day EEG monitoring.

The phenomenon of déjà vu (DV, "already seen") is an aberration of mental activity associated with the perception of surrounding reality with the impression that objects, contexts, and people seen for the first time are perceived for some period as already familiar. This phenomenon is a member of the group of derealization disorders, which also includes symptoms such as the "already experienced," "already heard," and "jamais vu" ("never seen") phenomena. The term was first used by the French psychologist E. Boirac in his book *L'Avenir des Sciences Psychiques*.

Particular interest in DV comes from the fact that this phenomenon is encountered in in healthy people (up to 97% of the population) spontaneously or in conditions of sleep disorders and anxiety. Until recently, it was not clear why some people never experience DV. Studies reported by Brazdil in 2012 [9] described interesting data obtained by magnetic resonance morphometry. Healthy people who have experienced DV, as compared with subjects who have never experienced it, had smaller volumes of gray matter in the mesotemporal areas on both sides (hippocampus, parahippocampal gyrus, insula, superior temporal gyrus, basal ganglia, thalamus), i.e., structures involved in generating DV. Decreases in the volume of gray matter correlated with the frequency of DV [10].

It is known that DV may be a sign of a number of mental and neurological diseases: Charles Bonnet syndrome, temporal epilepsy (TE), depression, and schizophrenia; DV is an early symptom of space-occupying brain lesions [3, 4, 9, 19].

Thus, there is a need to identify criteria for differential diagnosis, when DV is regarded as a variant of normal and when it is a symptom of disease.

The current literature contains many publications addressing the incidence of DV, the mechanisms by which it develops, and its clinical characteristics [5, 7, 10, 14, 19]. We have described the main characteristics of the phenomenon of DV useful for clinical differential diagnosis in healthy subjects [1], as well as in space-occupying brain lesions and epilepsy [2].

DV is of special interest in epilepsy. DV as aura occurs in 10% of patients with TE [13]. Autosomal dominant TE (ADTE) is characterized by focal seizures with auditory

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TABLE 1. Patterns on Routine Interictal EEG Recordings in Epilepsy with DV

EEG pattern	Number of patients	
	n	%
No pathological (epileptiform or slow-wave) activity	6	22.22
Focal slow-wave activity	4	14.81
Epileptiform signs – generalized and partial (sharp waves, multiple spikes, sharp wave-slow wave complexes)	12	44.44
Nonspecific changes (diffuse changes in bioelectrical activity with dysfunction of the median structures)	15	55.56

Note. Some patients showed combined EEG signs.

symptoms or aphasia. Mutations in the *LGI1* gene are seen in more than 50% of patients with ADTE. Patients with ADTE with mental episodes (DV and fear) have been identified more recently, without classical aphasia or auditory symptoms. These patients were found to carry a new mutation in the *LGI1* gene – Arg407Cys – which does not prevent secretion of the protein in vitro, as occurs with the previously described mutations [15, 16]. Studies of brain areas involved in DV were undertaken in patients with TE with and without the DV phenomenon [12]. A voxel-orientated analysis by 18FDG-PET was performed. Patients with TE and DV had ipsilateral foci of hypometabolism in the superior temporal gyrus and the parahippocampal region, in the immediate vicinity of the prerhinal and entorhinal cortex.

The main unanswered clinical question is that of whether the presence of DV is a sign that epilepsy is present. In 1983, Neppe (cited in [18]) noted a higher incidence of DV in patients with TE – 86%, as compared with 68% in the control group – though the difference was not statistically significant. Other authors [18] have taken the view that DV may represent an independent simple partial seizure in the structure of a complex partial seizure and that it is the aura for secondary generalized convulsive seizures.

This question probably cannot be answered without recording the specific ictal electroencephalogram patterns of DV, though we were unable to find any reports on EEG traces during the DV phenomenon in the literature available to us.

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

MATERIALS AND METHODS

A total of 166 subjects (mean age 25.17 ± 9.19 years; 63.2% women) took part in the study. The characteristics of DV were compared in two separate groups – group 1 consisted of healthy subjects (139 subjects) and group 2 consisted of patients with epilepsy (27 cases).

Paroxysmal manifestations of various types were excluded in all healthy subjects (syncopal states, autonomic paroxysms, psychogenic, epileptic, febrile seizures, etc.) throughout their lives. Each third subject was selected from this group for standard EEG investigations to exclude epileptiform activity.

Diagnoses in patients of group 2 were established using data from clinical neuroimaging and neurophysiological investigations.

The following investigation methods were used: 1. All subjects were interviewed using a specially developed original questionnaire to identify the characteristics of DV. The questionnaire was created on the basis of published data and were designed to identify the presence of DV, its frequency, duration, emotions, concomitants, and fear before onset of this derealization state. 2. Routine EEG. 3. Prolonged ambulatory EEG monitoring ("Holter EEG") for 12–18 h in 47 cases. EEG recordings were made only in the memory card of the recording device, in the automatic regime without phono- or photostimulation, with the subject entirely free and independent of a computer. This procedure was conducted in 20 healthy subjects and all epilepsy patients. This method allows patterns of rare seizures (particularly DV) to be identified during the patient's normal waking period. 4. Instrumented investigation methods: magnetic resonance and computerized tomography (MRI, CT), standard EEG for confirmation of diagnoses and clarification of the locations of lesion foci (space-occupying brain lesions, epileptic foci).

Data were analyzed statistically in Statistica 7.0 for Windows. Parametric and nonparametric statistical methods were used.

RESULTS

Assessment of EEG recordings from patients with epilepsy and the presence of DV in the clinical picture involved analysis of both routine EEG traces and ambulatory EEG monitoring.

Analysis of routine interictal EEG recordings (Table 1) showed that epileptiform signs and nonspecific changes to bioelectrical activity were seen with identical frequencies (44.4% and 55.5%, respectively). Patients with no EEG changes and slow-wave activity were significantly rarer.

As derealization disorders are, according to published data, "tied" to the right hemisphere, the hemisphere lateralization of epileptiform signs was analyzed in those patients

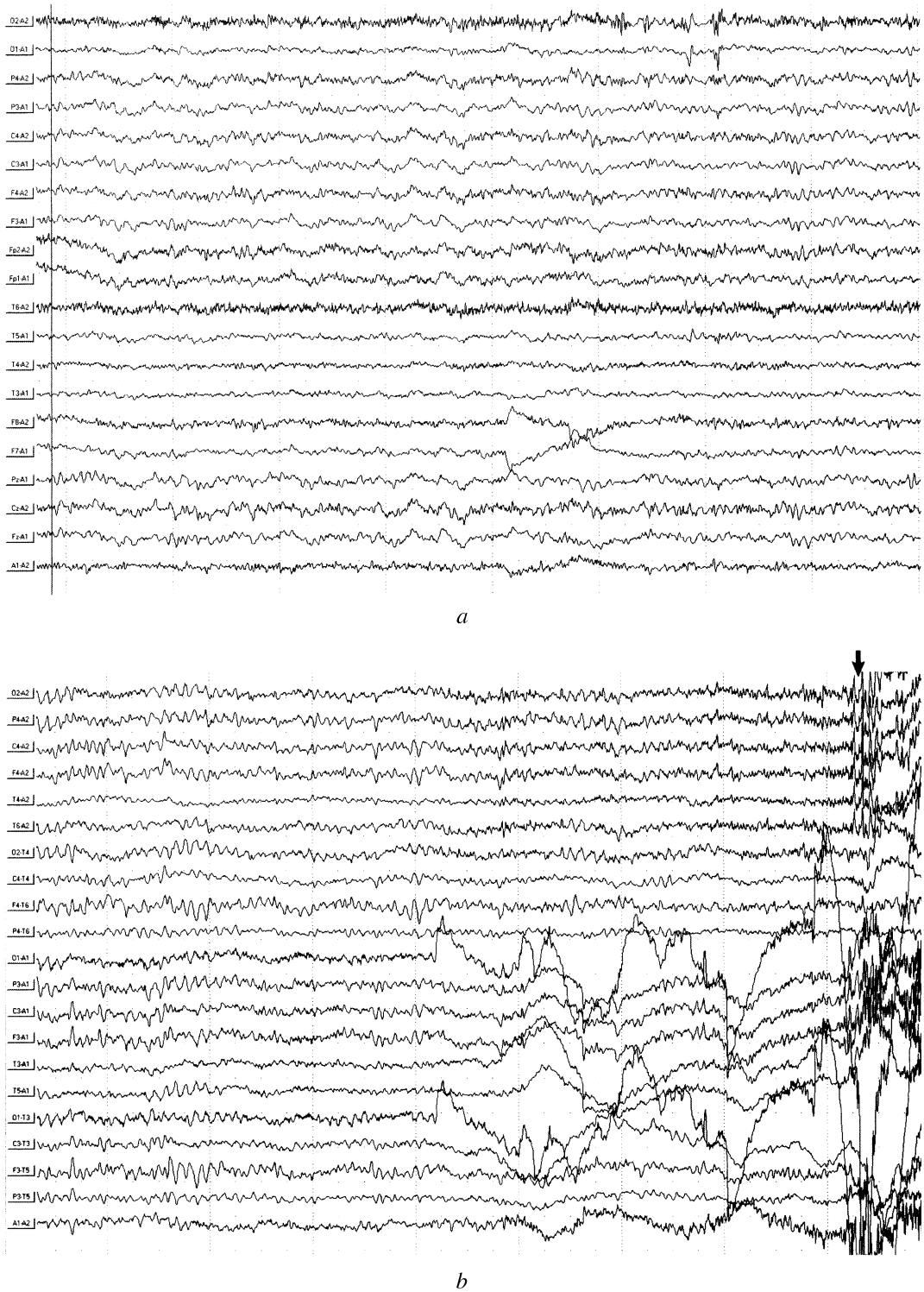

Fig. 1. EEG of patient D, aged 29 years. *a*) Baseline EEG; *b*) sec before patient marked DV (arrow); *c*) continuation of DV.

in whom they were seen. Contrary to expectation, routine EEG did not identify any hemisphere localization for pathological changes, which may be evidence that there is no connection between the hemisphere lateralization of interictal epileptiform activity on the EEG and the occurrence of the DV phenomenon.

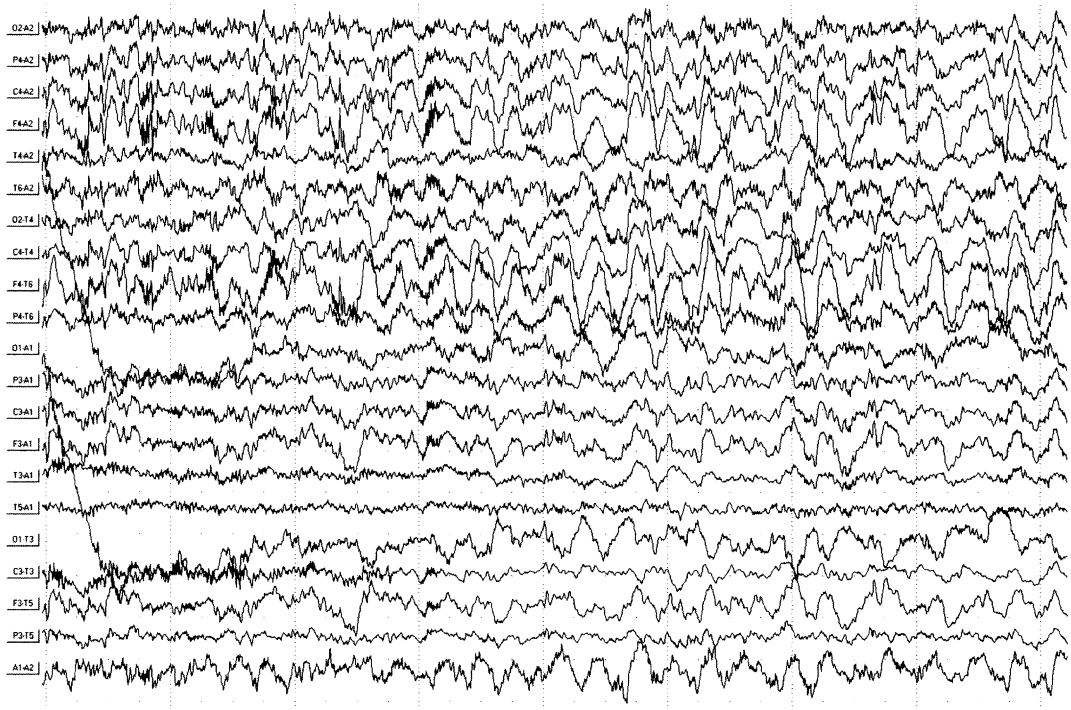

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Fig. 1. Continued.

The pattern of electrophysiological changes in derealization disorders was clarified using ambulatory EEG monitoring with simultaneous recording of the patient's feelings and states. During EEG monitoring, four patients showed signs of the DV phenomenon. We present descriptions of these patients' experiences below, along with their corresponding EEG changes.

Patient D, aged 29 years, driver. Raised and developed normally. From age 15 years, started to experience rare DV in the form of the "déjà vu" sensation lasting for periods of up to 5 sec. At age 24 years, the patient was involved in a road traffic accident and received a closed craniocerebral trauma (CCCT) with cerebral concussion. At 6–7 months post-injury, the patient started to experience generalized convulsive seizures with tongue-biting.

These episodes occurred at a rate of five per year. The incidence of "déjà vu" episodes increased, becoming more frequent and "clearer."

In April 2011, the patient was seen at the Scientific Center for Neurology, Russian Academy of Medical Sciences. By this time, seizure frequency was once per month, while the frequency of DV was 1–2 episodes per week, lasting up to 30 sec. The occurrence of DV was accompanied by fear and unpleasant sensations, though the patient nonetheless wanted to experience this state again. Brain CT scans did not identify any pathology. That EEG showed dysfunction of stem structures on the background of moderate diffuse changes in bioelectrical activity in the form of disorganized rhythms with some reduction in amplitude. Hyperventilation produced a degree of bilaterally synchronized paroxysmal activity, predominantly in the frontal-central areas, greater on the left.

The patient underwent 12-h EEG monitoring during which the "out of body" sensation (derealization) and anxiety occurred, followed by the "déjà vu" sensation, accompanied by an unpleasant feeling of melancholy. The duration of the DV episode was almost 20 sec. The following EEG changes were observed during this episode (Fig. 1).

The baseline EEG recording showed no paroxysmal activation. A skin galvanic reaction was detected a fraction of a second before the patient pressed the button (beginning of seizure),after which bilaterally synchronized multispike activity of amplitude up to 150 ∝V appeared, along with a large number of myogram artifacts. As the seizure developed, slow-wave activity in the θ–δ range appeared, with amplitude up to 200 ∝V, the greatest amplitude being seen in the right temporal leads. The total duration of slow-wave activity was 20 sec, after which the pathological rhythm was replaced by baseline activity.

Dipole localization using the BrainLock program showed that the foci of both initial and subsequent slow-wave multispike activity were located in the medial areas of the

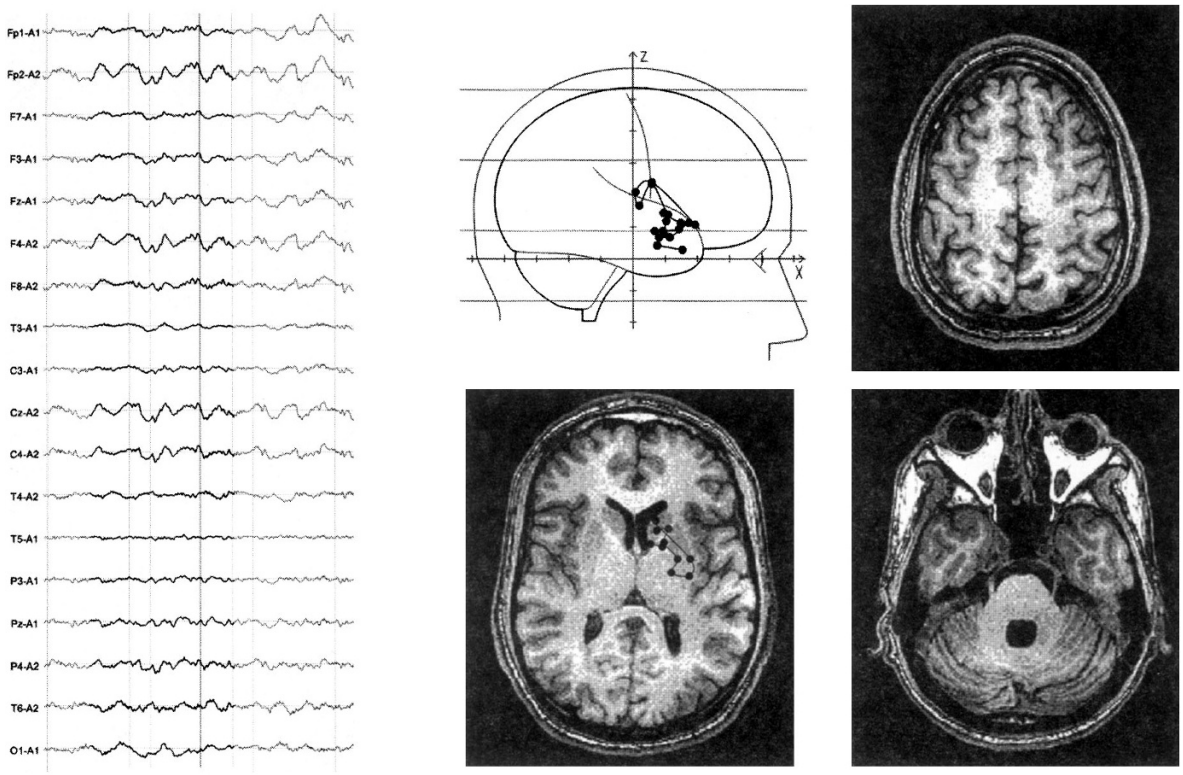

Fig. 2. Patient D. Location of focus of pathological slow-wave activity during the sensation of déjà vu. *a*) Fragment of the EEG analyzed; *b*) dipole location using the BrainLock program. Dots indicate the locations of activity superimposed on the standard MRI. Clear right-hemisphere lateralization is seen, with predominant activity in the medial parts of the right temporal lobe.

temporal and frontal lobes of the right hemisphere (Fig. 2). The other three ambulatory EEG recordings during DV in patients with epilepsy showed multispike activity for periods of 8 sec and were also characterized by clear right-hemisphere lateralization, with predominance in the temporal area.

We will now present observations of a healthy woman with the DV phenomenon.

Subject S, aged 20 years. She complained that for a long time (from age 15 years) she had experienced moderate tension headaches with a tendency to weather sensitivity. From about this same time, she had her first experiences of the "déjà vu" phenomenon, with a tendency to increase in frequency. Hypothymic states developed over the last two years, resulting in medical attendance.

On examination, complaints of headaches persisted, along with episodes of DV with a frequency of several per day and durations of up to 10 sec, accompanied by positive emotional coloration ("surprise, interest"), but no fear. Neurological status included symptoms of autonomic dysfunction of the parasympathetic type, in the form of acrohyperhidrosis. An MRI brain scan revealed no pathology; an ultrasound scan of the major arteries of the head found only age-related characteristics. The EEG showed moderate diffuse changes in brain bioelectrical activity, with no epileptiform signs.

During one-day ambulatory EEG monitoring, the patient noted an episode of a sensation similar to those previously experienced. There were feelings of present unreality and the patient thought she would experience DV and therefore pressed the button. She had interest in her state; and experienced pleasant feelings. There was as simultaneous feeling of "anticipation," of knowing what was going to happen next. This state lasted about 10–15 sec (Fig. 3).

As shown from the EEG fragment presented, epileptiform changes during the episode of DV were not seen in this healthy woman. The only change was desynchronization of the rhythm, evidencing the initially non-epileptic nature of DV in this healthy subject.

DISCUSSION

As discussed above, the main questions in studies of the phenomenon of DV relate to determination of its clinical significance, i.e., whether or not it is pathological and thus requires treatment; another is to identify the mechanism of its occurrence.

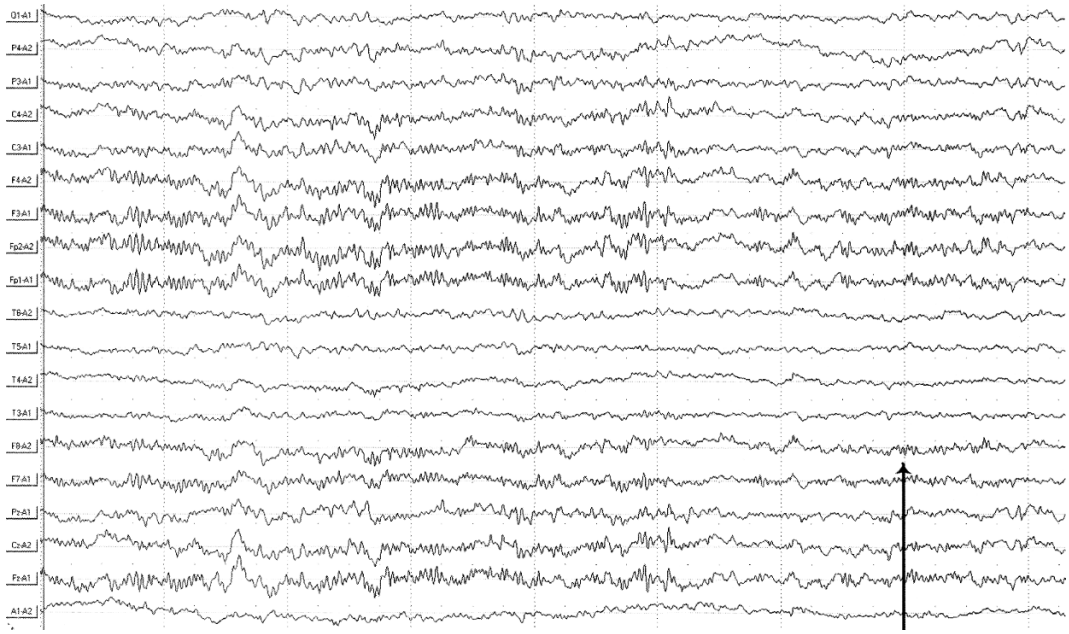

Fig. 3. EEG trace, subject S. Feeling of DV. The subject's marker is shown by the arrow. The EEG shows rhythm desynchronization. There are no epileptiform signs.

Most authors tend to the view that DV is not a psychopathological phenomenon, but a neurological status, and analog of epilepsy but with involvement of the psychoassociative zones [10].

An original hypothesis for the occurrence of DV was published by Spatt [14], who suggested that the functions of the hippocampus and prefrontal cortex include recognition of new information and relating this to previous experience. The parahippocampal system coordinates this operational during interruptions to its functioning, unfamiliar information seems familiar. The author concluded that DV occurs as a result of impairment to the contact of the neocortex with medial temporal structures on the background of decreased cortical influences (sleep, tiredness).

The episodes of DV recorded in this study were heterogeneous in terms of their origins. Considering the EEG data, along with the clinical, psychological, neuroimaging investigations, it can be suggested that there are two types of DV – pathological and nonpathological.

Most healthy subjects had nonpathological (nonepileptic) DV, which did not produce the epileptiform pattern on the EEG, had low frequency and duration, positive emotional coloration, and was generally induced by mental and physical overstress and lack of sleep. The possibility that nonpathological DV is not associated with impairments to neuron functioning and spontaneous neuron discharges cannot be completely excluded. However, propagation of this activity was probably so minor and local (in the mediobasal parts of the temporal lobe – the parahippocampal zone) that there is no clear pattern on the EEG. In epilepsy, the pathological, epileptic form of DV occurs, characterized by a specific pattern of activity on the EEG, with high frequency, long durations, and negative emotional coloration. Pathological DV occurs as a result of extreme neuron discharges of groups of cells and is, in essence, a partial psychogenic seizure.

The identification of two types of DV has also been supported by non-Russian authors, though without electrophysiological confirmation or grounding [6].

EEG data point to the involvement of the right hemisphere in generating DV. Some EEG traces made during DV are characterized by sharp-wave activity in the right hemisphere, lasting up to 8 sec, and a longer-lasting phenomenon consisting of the transition of sharp-wave to slow-wave activity. It may be that DV forms not in just one hemisphere, but as a result of impairments to the interaction between the two hemispheres. It is interesting to note that zones of hypometabolism have been seen [8, 11, 17] in PET and SPECT results in structures of the temporal lobe (entorhinal, prerhinal cortex). The slow-wave activity seen during the episode of DV in the patient with epilepsy may be an electrophysiological manifestation of hypoperfusion, as described previously.

Thus, the phenomenon of DV during epilepsy is characterized on the EEG by onset with multispike activity in the right temporal leads combined in some case (when the phenomenon lasts longer) with final slow-wave θ–δ activity in the right hemisphere. Complex clinical and electro-

physiological studies identified two types of DV: epileptic, typical of patients with epilepsy and equivalent to an epileptic seizure, and non-epileptic, typical of healthy people and in essence a psychological phenomenon.

REFERENCES

1. P. N. Vlasov and A. V. Chervyakov, "Significance of the phenomenon of déjà vu in healthy subjects," *Nevrol. Neiropsikh. Psikhosomat.*, No. 2, 53–57 (2009).
2. P. N. Vlasov, A. V. Chernyakov, S. V. Urakov, and A. A. Lukshina, "Diagnostic significance of the phenomenon of déjà vu in the clinical aspects of brain tumors," *Annaly Klin. Eksperim. Nevrol.*, No. 3, 26–31 (2011).
3. T. A. Dobrokhotova, S. V. Urakov, and T. A. Chebysheva, "Mental disorders in tumors of the cerebral hemispheres," in: *Neuropsychiatry*, Binom, Moscow (2006), pp. 107–131.
4. V. A. Karlov, "Current concepts in the treatment of epilepsy," *Zh. Nevrol. Psikhiat.*, **9**, No. 5, 4–7 (1999).
5. N. Adachi, N. Koutromanidis, R. D. C. Elwes, et al., "Interictal 18FDG PET findings in temporal lobe epilepsy with déjà vu," *J. Neuropsych. Clin. Neurosci.*, No. 11, 380–386 (1999).
6. N. Adachi, N. Akanuma, M. Ito, et al., "Two forms of déjà vu experiences in patients with epilepsy," *Epilepsy Behav.*, **18**, No. 3, 218–222 (2010).
7. J. Bancaud, R. Burnet-Bourgin, P. Chanvel, and E. Hargren, "Anatomical origin of déjà vu and vivid 'memories' in human temporal lobe epilepsy," *Brain*, **117**, 71–91 (1994).
8. F. Bartolomei, E. Barbeau, M. Gavaret, et al., "Cortical stimulation study of the role of rhinal cortex in déjà vu and reminiscences of memories," *Neurology*, **63**, No. 5, 858–864 (2004).
9. M. Brazdil, R. Marecek, T. Urbanek, et al., "Unveiling the mystery of déjà vu: The structural anatomy of déjà vu" *Cortex*, No. 1, 1–4 (2012).
10. A. S. Brown, "A review of the déjà vu experience," *Psychologic Bull.*, **129**, 394–413 (2003).
11. J. Engel, Jr., "The timing of surgical intervention for mesial temporal lobe epilepsy: a plan for a randomized clinical trial," *Arch. Neurol.*, **56**, 1338–1341 (1999).
12. E. Guedja, S. Aubert, A. McGonigal, et al., "Déjà vu in temporal lobe epilepsy: Metabolic pattern of cortical involvement in patients with normal brain MRI," *Neuropsychologia*. **48**, No. 7, 2174–2181 (2010).
13. A. Palmini and P. Gloor, "The localizing value of auras in partial seizures: a prospective and retrospective study," *Neurology*, **42**, 801–808 (1992).
14. J. Spatt, "Déjà vu: Possible parahippocampal mechanisms," *J. Neuropsychiatry Clin. Neurosci.*, **14**, 6–10 (2002).
15. P. Striano, A. Gambardella, A. Coppola, et al., "Familial mesial temporal lobe epilepsy (FMTLE): a clinical and genetic study of 15 Italian families," *J. Neurol.*, **255**, 16–23 (2008).
16. P. Striano, G. Busolin, L. Santulli, et al., "Familial temporal lobe epilepsy with psychic auras associated with a novel LGI1 mutation," *Neurology*, **76**, No. 13, 1173–1176 (2011).
17. Y. Takeda, T. Kurita, K. Sakurai, et al., "Persistent déjà vu associated with hyperperfusion in the entorhinal cortex," *Epilepsy Behav.*, **21**, No. 2, 196–199 (2011).
18. W. van Paesschen, M. D. King, J. S. Duncan, and A. Connelly, "The amygdala and temporal lobe simple partial seizures: a prospective and quantitative MRI study," *Epilepsia*, **42**, 857–862 (2001).
19. C. Warren-Gash and A. Zeman, "Déjà vu," *Practical Neurology*, No. 3, 1060–109 (2003).