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POTENTIALLY PATHOLOGICAL ALPHA-PATTERN AS A VARIANT OF VIGILANCE EEG IN DRUG-RESISTANT EPILEPSY

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As a result of pathomorphosis affecting the mechanisms of electrical activity generation interictal EEG may show reduced epileptiform changes whereas clinically apparent epileptic seizures may be present. In these cases patterns of dominant alpha activity are sometimes recorded on the scalp. In this study variations of alpha activity in patients with refractory epilepsy are classified. A group of 50 refractory epilepsy patients aged between 20 and 55 years who were submitted to Polenov Russian Scientific Research Institute of Neurosurgery in 2014-2017 was included in this study. They underwent scalp EEG as a part of their presurgical assessment. In 12 cases patterns of potentially pathological alpha activity were observed. Three variations of alpha-patterns were described: 1) alpha-rhythm with decreased regional diversity and a marked synchronization in temporal areas; 2) alpha-rhythm with reduced epileptiform complexes integrated into the spindles, 3) decelerated non-rhythmic alpha activity distorted by the higher frequency components. Distinguished varieties of potentially pathological alpha-activity according to their order here represent gradual functional decline of normal thalamo-cortical interaction. Considering clinical manifestation of drug-resistant epilepsy with frequent seizures in these patients, reported varieties of alpha activity can not be interpreted as Landolt's syndrome (forced normalization of EEG). Invasive electrocorticographic monitoring demonstrated that bursts of sharpened polyphasic waves coinciding with alpha-rhythm on scalp EEG are consistent with epileptic discharges on the brain cortex surface. This allows to think of these components as correlates of epileptic activity. Therefore, on a number of occasions in patients with epilepsy a dissonance between clinical signs and electroencephalographic patterns recorded during restful wakefulness may be observed, when epileptiform components are absent or reduced to nonspecific complexes.

Keywords: epilepsy; electroencephalography (EEG); alpha-rhythm; video-EEG monitoring; electrocorticography.

УСЛОВНО-ПАТОЛОГИЧЕСКИЙ АЛЬФА-ПАТТЕРН КАК ВАРИАНТ ЭЭГ БОДРСТВОВАНИЯ ПРИ ФАРМАКОРЕЗИСТЕНТНОЙ ЭПИЛЕПСИИ

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В результате патоморфоза механизмов генерации биоэлектрической активности при развернутой клинической картине эпилепсии на интериктальной ЭЭГ возможна редукция эпилептиформных изменений. В таких случаях может регистрироваться паттерн с доминирующей альфа-активностью. Целью исследования являлась систематизация вариантов альфа-активности у пациентов с фармакорезистентной эпилепсией. Обследована группа из 50 пациентов с фармакорезистентной эпилепсией в возрасте от 20 до 55 лет, находившихся на лечении в клинике Российского нейрохирургического института им. проф. А.Л. Поленова в 2014–2017 гг. В 12 случаях были зафиксированы паттерны условно-патологической альфа-активности. Были выделены три основных варианта

альфа-паттернов: 1) альфа-ритм со снижением зональных различий и выраженной синхронизацией в височных отделах; 2) альфа-ритм с включением в структуру «веретен» редуцированных эпилептиформных комплексов; 3) замедленная неритмизированная альфа-активность, искаженная высокочастотной составляющей. Выделенные варианты условно-патологической альфа-активности в представленной очередности отражают поэтапное нарастание дисфункции таламо-кортикальных взаимоотношений. Учитывая имеющуюся у пациентов фармакорезистентную эпилепсию с частыми приступами, зафиксированные варианты альфа-активности не могут рассматриваться как проявления синдрома насильственной нормализации (синдрома Ландольта). Инвазивный мониторинг биоэлектрической активности коры показал, что вспышки заостренных полифазных волн в структуре альфа-ритма на скальповой ЭЭГ совпадают с эпилептическими разрядами на коре, что позволяет считать такие элементы коррелятами эпилептической разрядной активности на коре. Таким образом, в ряде случаев у больных с эпилепсией может наблюдаться «диссоциация» клинических проявлений и электроэнцефалографической картины в состоянии пассивного бодрствования, когда эпилептиформные элементы могут полностью отсутствовать или редуцироваться до неспецифических комплексов.

Ключевые слова: эпилепсия; электроэнцефалография (ЭЭГ); альфа-ритм; видео-ЭЭГ-мониторинг; электрокортикография.

INTRODUCTION

Presently, elucidating epilepsy pathogenesis and improvement of its diagnostics and treatment are among the most significant challenges in neurology [7]. Epilepsy is a relatively widespread condition, with a worldwide incidence rate of 5–10 per 1000 people. In the Russian Federation, the estimated incidence of epilepsy is 1.2–7.2 per 1000 people [1]. Since the disease is often diagnosed late, it results in the development of drug-resistant epilepsy observed in 20%–30% of all cases.

For many years, electroencephalography (EEG) remains the leading diagnostic method for patients with epilepsy [3, 6, 7], as a majority of patients experiencing regular epileptic seizures exhibit epileptiform activity on EEG. However, up to 30% of patients suffering from epileptic seizures and receiving antiepileptic therapy have only alpha activity with no epileptiform patterns on EEG during passive wakefulness [6]. This can be partially explained by changes in bioelectric activity with the involvement of critical thalamic mechanisms of alpha rhythms' generation [2, 12] as well as deformation and reduction of epileptiform changes, primarily induced by antiepileptic therapy. While the detection of pronounced alpha spindles on EEG indicates well-established adaptive mechanisms for upward and downward regulation, their disappearance demonstrates impaired sensorimotor integration [4]. Nonetheless, alpha range potentials on EEG do not always indicate well-being as they can be detected, for example, in a coma [16, 17]. The detection of alpha activity without epileptiform patterns on EEG significantly reduces the probability of diagnosing epilepsy, assessing its dynamics, and choosing a correct treatment regimen. Recent studies investigating alpha activity in patients with epilepsy have revealed only a reduction of variability in the frequency of the basic rhythm in these patients [15]. However, despite the apparent role of thalamocortical interactions in spreading epileptic discharges in experimental and mathematical models [2, 9, 20], its pathophysiological aspects are poorly understood to establish unambiguous relationships [21]. The characteristics of the alpha rhythm and its evolution are currently investigated in the normal EEG and non-epileptic pathological EEG using novel sophisticated methods of data processing [10, 19, 22]. A detailed analysis of alpha activity in patients with epilepsy receiving antiepileptic drugs (AEDs) will probably enhance the predictive value of EEG for these patients [11].

This study *aimed* to assess and systematize EEG patterns with a prevalence of alpha activity in patients with drug-resistant epilepsy.

MATERIAL AND METHODS

We analyzed the results of comprehensive clinical, neurophysiological, and neuroimaging examination of 50 patients with drug-resistant epilepsy aged between 20 and 55 years. Patients were examined and treated at the Clinic of the Polenov Russian Neurosurgical Institute during 2014–2017. The inclusion criteria in this study were as follows: (1) have focal drug-resistant epilepsy according to the International League Against Epilepsy (ILAE) criteria (treatment with two or more modern AEDs for more than 2 years); and (2) have stable rhythmic alpha activity on EEG during passive wakefulness.

Neurophysiological examination included conventional EEG with functional tests and long-term video-EEG monitoring during wakefulness and sleep. The surface EEG was recorded in standard leads using monopolar and bipolar montage with electrode locations according to the International 10–20 System.

In this study, 12 patients underwent long-term invasive neurophysiological monitoring to verify the location of the epileptic focus. Electrocorticogra-

phy (ECoG) was recorded using subdural electrodes in a bipolar montage; the electrodes were introduced through the burr holes. In addition, conventional EEG was recorded in parallel with ECoG to assess the distribution of bioelectric activity. The amplifier's bandwidth was 0.5–70 Hz in both variants.

Furthermore, neurophysiological examinations were performed using two diagnostic systems, "Mitsar-EEG-201" and "Mitsar-EEG-202."

RESULTS

All study participants have had epilepsy for a long time (3–29 years). The age at the disease onset varied between 1 and 51 years. The antiepileptic therapy included at least two modern AEDs, and plasma concentration of the drugs was within the therapeutic reference ranges. Thus, all patients fulfilled the ILAE criteria for drug-resistant epilepsy.

In this study, 22 of 50 patients had concordant clinical, neuroimaging, and neurophysiological findings. In addition, 10 patients exhibited a high index of stable regional epileptic activity along with alpha activity registered by EEG. In 12 patients, both conventional EEG and video-EEG monitoring during passive wakefulness demonstrated only stable alpha activity without any epileptic patterns. Bilateral epileptiform activity was registered in 26 patients using the surface EEG. Furthermore, 14 patients required two or three additional procedures of long-term video-EEG monitoring with a mean duration of 5.5 ± 2.2 h to verify the location of the epileptic area. In 13 patients, the sensitivity of video-EEG monitoring was insufficient to identify the location of the epileptic focus; these patients additionally underwent invasive monitoring of the brain bioelectrical activity.

Thus, conditionally pathological alpha activity on EEG was observed in 12 of 50 patients with drug-resistant epilepsy (Table 1). Of these, 8 patients developed epileptic seizures in childhood at the age of 3–16 years.

Table 1 shows that we failed to find significant correlations between the clinical manifestations, neuroimaging findings, and the type of EEG changes. However, we observed some deviations from the normal alpha activity during passive wakefulness.

The detected alpha activity differed from the standard alpha activity during passive wakefulness. We identified the following three main variants of conditionally pathological alpha activity (Figures 1–3):

1) alpha rhythm with reduced zonal differences and pronounced synchronization in the temporal areas;
2) alpha rhythm with reduced epileptiform elements within the spindles; and
3) slow non-rhythmic alpha activity distorted by high-frequency oscillations (sharpened waves).

Figure 2 shows a pattern of the background activity with well-modulated alpha rhythm. Regional high-amplitude sharpened oscillations are registered in the structure of alpha spindles. Usually, such findings are considered as a nonspecific pathological activity. Figure 3 shows weakly modulated sharpened alpha rhythm characterized by unstable frequency (8–11 Hz) and medium amplitude (up to 60–70 μV). Alpha activity prevailed in the central and temporoparietal leads. Asynchronous sharpened transients originating primarily from posteriotemporal areas were occasionally registered on EEG.

We described a clinical case of symptomatic temporal lobe epilepsy in a 40-year-old female patient with complex focal seizures to complete the classi-

Table 1

The characteristics of patients with predominant alpha activity on EEG during wakefulness

Таблица 1

Характеристика группы пациентов с доминирующей альфа-активностью на ЭЭГ бодрствования

No.	Gender/ age	Age at disease onset	Disease duration	Type of seizures	MRI	AEDs
1	Male/35	15	20	Partial sensory; complex: dialeptic, automotor	Moderate encephalopathy of mixed origin	Carbamazepine, Sodium Valproate, Phenytoin
2	Male/40	25	15	Partial: sensorimotor, vegetative; automotor	Hippocampal asymmetry ($L > R$), FCD	Perampanel, Carbamazepine
3	Male/36	19	17	Partial: sensory, vegetative; automotor	Pilocytic astrocytoma in the right lateral ventricle	Sodium Valproate, Levetiracetam

End of the table 1
Окончание табл. 1

No.	Gender/ age	Age at disease onset	Disease duration	Type of seizures	MRI	AEDs
4	Female/35	13	22	Partial: vegetative sensory, motor; complex: dialeptic, automotor; SGS	Right hippocampal sclerosis, heterotopia at the left lateral ventricle, cystic atrophy of the right frontal lobe	Sodium Valproate, Carbamazepine
5	Female/31	16	15	Complex partial vegetovisceral: dialeptic, automotor; SGS	Right hippocampal cyst, external hydrocephalus substitution	Sodium Valproate, Oxcarbazepine
6	Female/40	9	31	Partial psychosensory; complex automotor; SGS	Right hippocampal sclerosis	Sodium Valproate, Oxcarbazepine, Lacosamide, Levetiracetam
7	Female/27	24	3	Partial, psychomotor, SGS	Right hippocampal sclerosis, heterotopia	Sodium Valproate, Levetiracetam
8	Female/31	9	22	Partial: vegetative, psychomotor; GS complex automotor	Moderate mixed hydrocephalus	Sodium Valproate, Lacosamide, Levetiracetam
9	Male/32	12	20	Partial, vegetovisceral; GS	No pathological changes	Lacosamide, Levetiracetam
10	Female/30	26	4	Complex focal seizures with motor and ambulatory automatisms, rare GS	Bilateral subependymal heterotopia of the gray matter	Sodium Valproate, Levetiracetam, Oxcarbazepine
11	Female/30	3	27	Serial focal motor seizures, aura	No pathological changes	Zonisamide, Lacosamide
12	Male/31	11	20	Complex focal seizures	No pathological changes	Carbamazepine, Lacosamide, Levetiracetam

Abbreviations: AEDs, antiepileptic drugs; SGS, secondarily generalized seizures; GS, generalized seizures; MRI, magnetic resonance imaging; FCD, focal cortical dysplasia

fication of EEG patterns. The first seizure occurred at the age of 29 during delivery; she lost consciousness and developed motor automatisms (scratching movements of the hands and leg extension) with subsequent postictal confusion. After delivery, seizures continued with a frequency of 10–15 every month. The patient received several AEDs, including valproates, topiramate, trileptal, and vimpat; however, treatment regimens were changed several times because of ineffectiveness. Until hospitalization, the patient had 1–2 serial (4–5 per day) seizures per month with a duration of 2–3 min. Her treatment regimen included levetiracetam and lamotrigine. While video-EEG monitoring demonstrated two foci of pathological activity (sharpened waves) in the right and left temporoparietal areas, MRI brain scans revealed focal changes with possible vascular origin. In addition, we detected hippocampal asymmetry (D < S). Positron emission tomography/computed tomography (PET/CT) scanning revealed signs of glucose hypometabolism in the cortex of the right temporal lobe, primarily in the temporal pole and in the temporoparietal area of the left hemisphere. Owing to the partial concordance of clinical, neuroimaging, and neurophysiological findings, the patient underwent invasive monitoring to verify the location of the epileptic focus. Accordingly, subdural strip electrodes were placed bilaterally over the temporal lobes, and a dominant focus of epileptic ac-

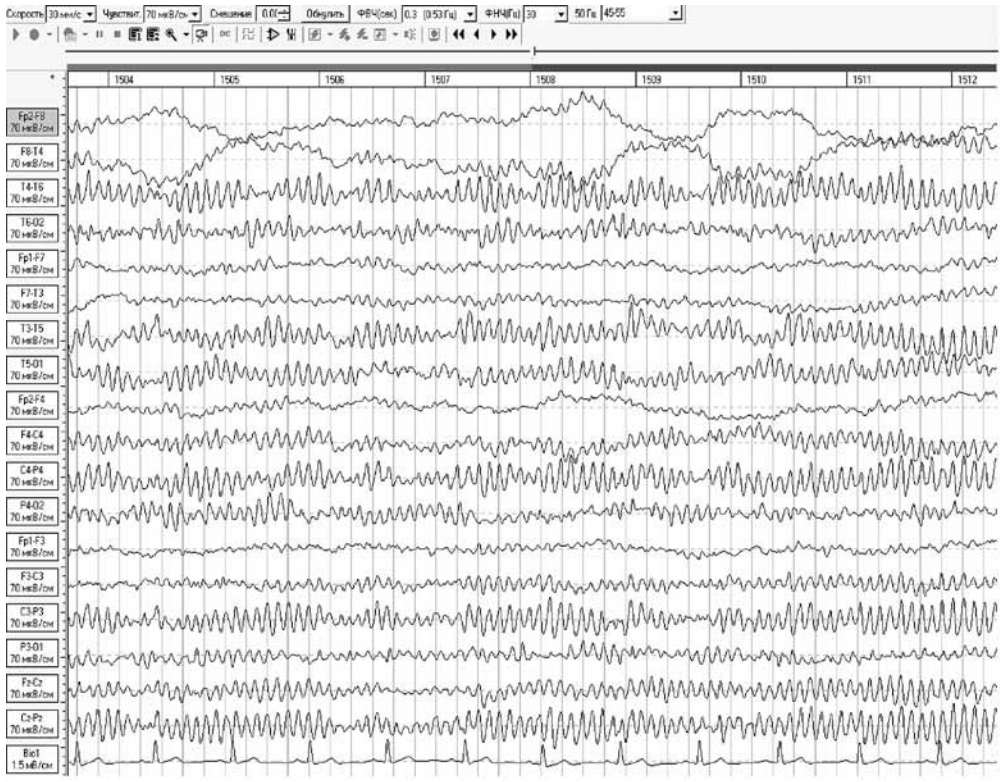

Fig. 1. Alpha rhythm with reduced zonal differences and pronounced synchronization in the temporal areas in a 36-year-old male patient with drug-resistant epilepsy

Рис. 1. Альфа-ритм со снижением зональных различий и выраженной синхронизацией в височных отделах. Больной К., 36 лет, фармакорезистентная эпилепсия

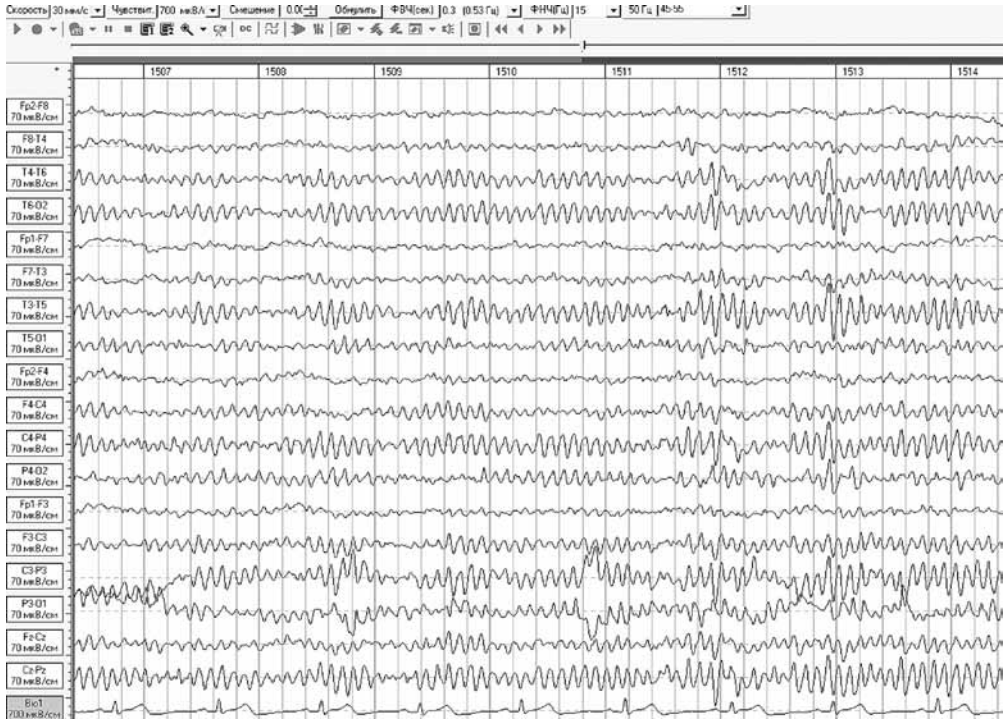

Fig. 2. Alpha rhythm with reduced epileptiform elements within the spindles in a 28-year-old female patient with drug-resistant epilepsy

Рис. 2. Альфа-ритм с включением в структуру «веретен» редуцированных эпилептиформных комплексов. Больная О., 28 лет, фармакорезистентная эпилепсия

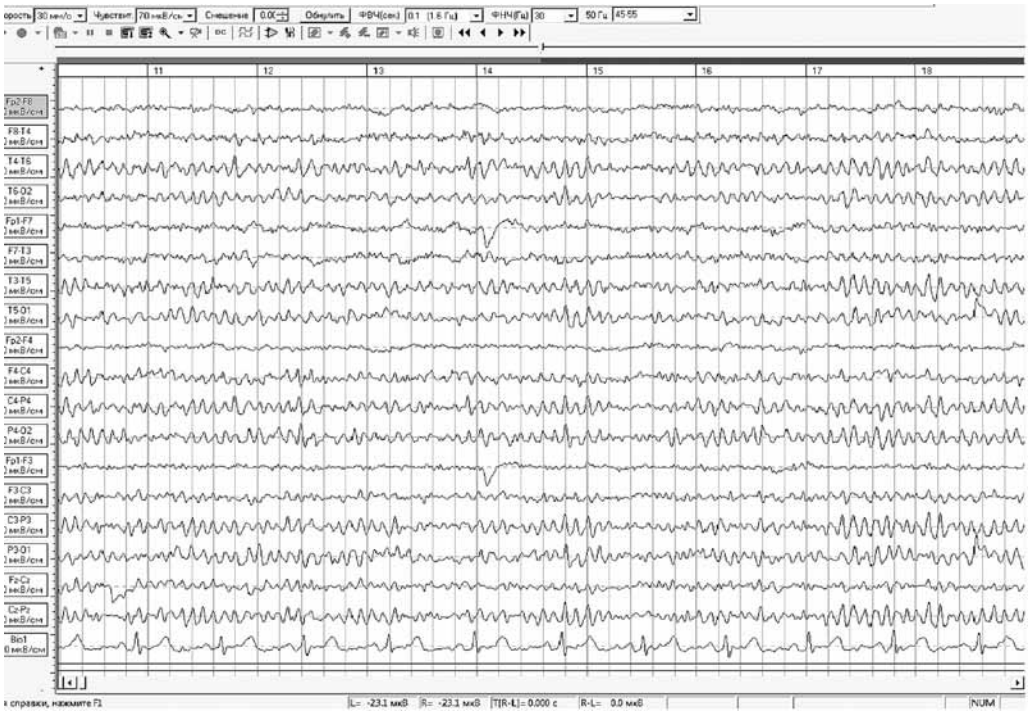

Fig. 3. Slow alpha activity distorted by rare high-frequency oscillations (sharpened waves) in a 38-year-old male patient with drug-resistant epilepsy

Рис. 3. Замедленная деформированная альфа-активность, искаженная высокочастотной составляющей, с редкими «заостренными» потенциалами. Больной С., 38 лет, фармакорезистентная эпилепсия

tivity (with activity index of 50%–60%) was detected in the right temporal lobe.

Hence, video-EEG monitoring with sleep deprivation did not allow identification of the exact location of a dominant epileptic focus. The pattern of the background activity included the alpha rhythm with abnormal zonal distribution, and diphasic and triphasic sharpened waves were occasionally detected.

Furthermore, we performed long-term invasive monitoring of the cortical bioelectric activity to verify the location of the epileptic focus. Strip electrodes were introduced through the burr holes and placed on the cortical surface of the temporal lobes in the right and left hemisphere under general anesthesia. After 2 days, we performed co-registration of the surface EEG and ECoG. The invasive monitoring revealed that diphasic and triphasic sharpened waves in the alpha rhythm coincided with epileptic discharges in the cortex (Figure 4). Our results suggested that sharp polyphasic waves in the structure of alpha spindles correlate with epileptic discharges in the cortex.

Finally, we detected an epileptic focus in the right temporal area. The patient underwent surgical removal of two-thirds of the temporal lobe, amygdala, and anterior hippocampus under electrophysiological control. The patient was examined after 1 year of surgery. Although no seizures occurred during this time, the patient had several episodes of aura. Video-EEG monitoring demonstrates steady alpha rhythm with no signs of focal or diffuse epileptiform activity.

DISCUSSION

We believe that the described variants of conditionally pathological alpha activity (in order of their appearance in the text) reflect gradually increasing dysfunction in thalamocortical interactions [2, 16].

The detected changes in the alpha rhythm do not necessarily indicate typical epileptiform activity. However, if a patient presents epileptic seizures with no response to therapy, these EEG changes can be considered conditionally pathological.

The presence of sharpened waves within normal or distorted alpha rhythm is probably explained by the reduction (incomplete conduction) of epileptiform activity from the cortex to the scalp surface because of the signal scattering in the dielectric structures of the skull and integumentary tissues [8]. Cortical epileptiform discharges are detected on the scalp only when they reach specific amplitude [3, 6, 7, 18]. In this description of alpha activity patterns, the focus should be on the relatively high frequency of EEG alpha patterns in patients diagnosed with epilepsy, especially in those with drug-resistant forms

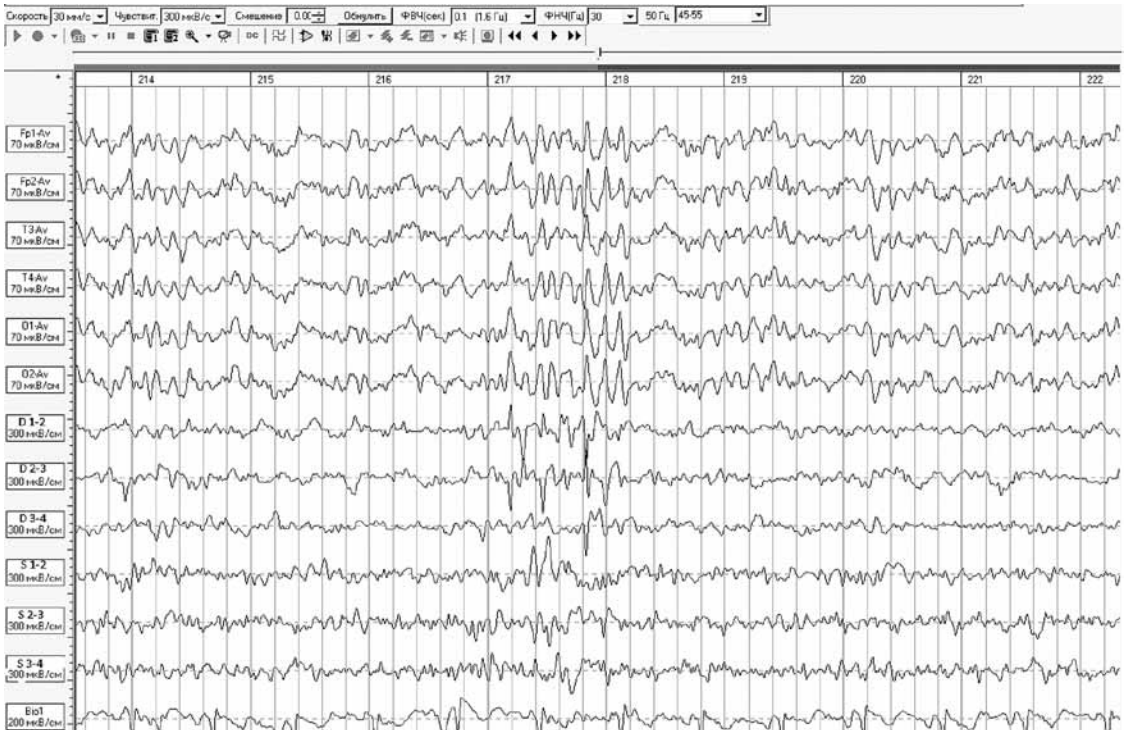

Fig. 4. Reduced cortical epileptiform activity on the surface EEG: results of invasive monitoring. Surface EEG signals were registered from the leads Fp1, Fp2, T3, T4, O1, and O2. Leads D and S correspond to corticographic strip electrodes in the right and left hemisphere, respectively. The Bio1 lead – ECG

Рис. 4. Редукция корковой эпилептической активности на скальповой ЭЭГ: результаты инвазивного мониторинга. Отведения Fp1, Fp2, T3, T4, O1, O2 скальповой ЭЭГ. Отведения D и S соответствуют кортикографическим стрип-электродам правого и левого полушария соответственно. Отведение Bio1 — ЭКГ

and frequent seizures. In this case, the clinical manifestations hinder the interpretation of the alpha pattern as a sign of Landolt syndrome (syndrome of forced normalization).

A hypersynchronous sharpened alpha rhythm has already been described as an equivalent of cortical epileptiform discharges [5]. These oscillations are similar to typical epileptiform discharges (sharp waves) when registered directly in the cortex. Some researchers consider sharpened theta/alpha activity with an average frequency of 9.5 Hz and increasing amplitude as one of the EEG patterns typical of the seizure beginning [14]. However, such EEG patterns are currently described as nonspecific pathological changes or lowered seizure threshold in most cases. Limited studies emphasize the importance of high-frequency oscillations in the alpha rhythm for detecting epileptogenic areas in the cortex [13].

Thus, patients with epilepsy often have discordance between the clinical manifestations and EEG findings during passive wakefulness. Perhaps, epileptiform discharges could be completely absent or reduced to nonspecific complexes in the structure of the basic rhythm.

CONCLUSIONS

1. Twelve of 50 patients with drug-resistant epilepsy had conditionally pathological alpha activity on EEG. Of these, eight developed epileptic seizures in childhood at the age of 3–16 years.
2. We described three main variants of conditionally pathological alpha activity as follows: (1) alpha rhythm with reduced zonal differences and pronounced synchronization in the temporal areas; (2) alpha rhythm with reduced epileptiform elements within the spindles; and (3) slow non-rhythmic alpha activity distorted by high-frequency oscillations (sharpened waves).
3. The described variants of conditionally pathological alpha activity reflect gradually developing dysfunction in thalamocortical interactions. The detected changes in the alpha rhythm do not necessarily indicate typical epileptiform activity. However, if a patient has epileptic seizures with no response to therapy, these EEG changes can be considered conditionally pathological.

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