Impact Factor: 5.723

ISSN: 2181-0982
DOI: 10.26739/2181-0982
www.tadqiqot.uz

JNNR

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH

VOLUME 4, ISSUE 5

2023

ISSN 2181-0982

Doi Journal 10.26739/2181-0982

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

ТОМ 4 НОМЕР 5

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH

VOLUME 4, ISSUE 5

ТОШКЕНТ-2023

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

Бухарский государственный медицинский институт и tadqiqot.uz

Рецензируемый научно-практический журнал “Журнал неврологии и нейрохирургических исследований”
Публикуется 6 раза в год
№5 (04), 2023
ISSN 2181-0982

Адрес редакции:
ООО Tadqiqot город Ташкент, улица Амира Темура пр.1, дом-2.
web: http://www.tadqiqot.uz/;
Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000

Макет и подготовка к печати проводились в редакции журнала.

Дизайн - оформления:
Хуршид Мирзахмедов

Журнал зарегистрирован в Управлении печати и информации г. Ташкента Рег. № от 01.07.2020 г.

“Неврологии и нейрохирургических исследований” 5/2023

Электронная версия журнала на сайтах:
https://tadqiqot.uz
www.bsmi.uz

MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN PUBLIC ASSOCIATION OF NEUROLOGISTS OF UZBEKISTAN

JOURNAL OF NEUROLOGY AND NEUROSURGICAL RESEARCH

Bukhara State Medical Institute and tadqiqot.uz

Peer-reviewed scientific and practical journal "Journal of Neurology and Neurosurgical Research"
Published 6 times a year
#5 (04), 2023
ISSN 2181-0982

Editorial address:
Tadqiqot LLC the city of Tashkent, Amir Temur Street pr.1, House 2.
Web: http://www.tadqiqot.uz/;
Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

Layout and preparation for printing held in the editorial office of the journal.

Design – pagemaker:
Khurshid Mirzakhmedov

Journal is registered at the Office of Press and Information Tashkent city, Reg. No. July 1, 2020

"Neurology and neurosurgical research" 5/2023

Electronic version of the Journal on sites:
www.tadqiqot.uz,
www.bsmi.uz

СОДЕРЖАНИЕ | CONTENT

ISSN: 2181-0982
www.tadqiqot.uz
JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH
ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

УДК: 616.821-4578/9012.56

Клычева Раушания Ислямовна
Андижанского государственный медицинский институт
Рахимбаева Гульнара Саттаровна
Ташкентская медицинская академия

ВИДЫ ЭПИЛЕПТИЧЕСКИХ ПРИСТУПОВ У ПАЦИЕНТОВ С ФОКАЛЬНОЙ СТРУКТУРНОЙ ЭПИЛЕПСИЕЙ В ЗАВИСИМОСТИ ОТ ЛОКАЛИЗАЦИИ И ЛАТЕРАЛИЗАЦИИ ЭПИЛЕПТОГЕННОГО ФОКУСА (ЛИТЕРАТУРНЫЙ ОБЗОР)

http://dx.doi.org/10.5281/zenodo.10454050

АННОТАЦИЯ

Знание семиотики эпилептических приступов, возникающих как результат нарушений в различных долях головного мозга, имеет существенное клиническое значение в первую очередь для выявления эпилептогенных структурных поражений головного мозга и/или в рамках предоперационного обследования у пациентов с резистентной эпилепсией

Ключевые слова: эпилепсия, эпилиоптогенный очаг, латеризация, эпилептические приступы

Klycheva Raushaniya Islomovna
Andijon davlat tibbiyot instituti
Rahimbayeva Gulnara Sattarovna-
Toshkent tibbiyot akademiyasi

EPILEPTOGEN FOKUSNING LOKALIZATSIYASI VA LATERALIZATSIYASIGA QARAB FOKAL STRUKTURAVIY EPILEPSIYA BILAN OG'RIGAN BEMORLARDA EPILEPTIK HURUJLAR TURLARI (ADABIYOTLAR SHARHI)

ANNOTATSIYA

Miyaning turli loblarida buzilishlar natijasida yuzaga keladigan epileptik tutilishlarning semiotikasini bilish, birinchi navbatda, epileptogen strukturaviy miya lezyonlarini aniqlash va/yoki chidamli epilepsiya bilan og'rigan bemorlarda operatsiyadan oldingi tekshiruvning bir qismi sifatida muhim klinik ahamiyatga ega

Kalit so'zlar: epilepsiya, epilioptogen focus, laterlizatsiya, epileptic hurujlar

---

Klycheva Raushaniya Islyamovna
Andijan State Medical Institute
Rakhimbayeva Gulnara Sattarovna
Tashkent Medical Academy

CORRELATION OF THE DEGREE OF COGNITIVE IMPAIRMENT IN DIFFERENT FORMS OF EPILEPSY WITH BLOOD CORTISOL LEVELS (LITERATURE REVIEW)

ANNOTATION

Knowledge of the semiotics of epileptic seizures arising as a result of abnormalities in different lobes of the brain is of significant clinical importance primarily for the detection of epileptogenic structural lesions of the brain and/or as part of preoperative examination in patients with intractable epilepsy.

Keywords: epilepsy, epilioptogenic focus, laterisation, epileptic seizures

---

Introduction. An epileptic seizure is a transient clinical manifestation of pathological excessive or synchronous neural activity in the brain (conceptual definition of an epileptic seizure, ILAE, 2005). Seizures do not always mean that a person has epilepsy unless the criteria for diagnosing epilepsy are met. As there are a number of conditions that may be associated with paroxysmal events that can mimic seizures/epilepsy, they should be carefully ruled out.

Self-limiting childhood epileptic syndromes or devastating infantile epileptic encephalopathies are rarely seen in persons over 18 years of age. Using electroencephalographic video monitoring, frontal seizures were able to be categorised into 6 groups: 1) focal clonic motor; 2) asymmetric tonic; 3) hyperkinetic (psychomotor, hypermotor, complex partial); 4) opercular; 5) absences; and 6) seizures resembling medial temporal lobe epilepsy. Temporal lobe epilepsies can be divided into 2 types according to well-defined syndromes: 1) medial, or amygdalo-hippocampal, associated with lesions of the limbic system in the deep regions of the temporal lobe; 2) lateral, or neocortical, based on lesions of the temporal neocortex. Classic symptoms of parietal epilepsy are represented by paresthesias, usually contralateral to the seizure focus. Patients may describe ictal somatosensory symptoms as "goosebumps

crawling", "numbness", "tingling", "twitching". In occipital epilepsy, ictal visual symptoms may be either positive in the form of "bright flashes", "circles", "goosebumps", "birds", "flickering bright spots", etc., or negative in the form of "darkness before the eyes" (amaurosis) or "black spots" (black scotomas). Various oculomotor symptoms, such as tonic eyeball retraction, are common in occipital seizures. On outpatient appointment, self-limiting epileptic seizures characteristic of childhood syndromes or devastating infantile epileptic encephalopathies are rarely seen in persons over 18 years of age [1]. Here are the main data on the epileptic syndromes most commonly encountered in adult practice.

Frontal lobe epilepsy

In this form of epilepsy, foci of epileptogenic activity occur in the frontal lobes, including the orbitofrontal, frontopolar, dorsolateral, operculum, motor and supplementary motor areas, or in the cingulate gyrus.

The previously seemingly chaotic variety of ictal manifestations of frontal seizures, thanks to the introduction of electroencephalographic video monitoring into widespread clinical practice, has made it possible to classify paroxysms originating from the frontal lobe into 6 groups: 1) focal clonic motor; 2) asymmetric tonic; 3) hyperkinetic seizures - HP (psychomotor, hypermotor, complex partial); 4) opercular; 5) absences; 6) paroxysms resembling medial temporal lobe epilepsy.

Focal clonic motor seizures

Focal clonic motor seizures, manifested in isolation, are not accompanied by loss of consciousness and reflect ictal activity in the primary motor area. This type of seizure is characteristic of various frontal and extrafrontal epileptic syndromes leading to secondary activation of the primary motor cortex.

Seizures are rhythmic contractions and relaxations of muscles that may be localised or spread to other parts of the body ("Jacksonian march"). The duration of an episode rarely exceeds 1-2 min. After its termination, Todd's palsy is often observed, the localisation of which is of great informational value (the palsy is usually contralateral to the area of seizure onset). Electroencephalogram (EEG) in the postictal period may show local slowing, and neuroimaging may show transient local brain oedema [1,3].

Asymmetric tonic seizures

Asymmetric tonic seizures are caused by excitation of the secondary sensorimotor zone. Episodes are short (10-40 s) and most often consist of a bilateral asymmetric increase in arm tone with withdrawal/adduction and raising/lowering and bending at the elbow joints without disturbance of consciousness. Less frequently athetoid movements in the contralateral arm, leg or half of the face, kicking, pedalling or pacing movements in the legs, their tonic or dystonic setting are observed. The seizure may be preceded by an aura in the form of tingling, numbness, or tension. The tonic phase of the seizure may begin with involuntary vocalisation, and there may be a lack of speech after the seizure. Post-ictal confusion is very rare. Asymmetric tonic seizures are characterised not only by positive (tonic) but also negative (atonic) motor manifestations. Helastic seizures have also been described, arising when epileptogenic foci are localised in the brain region in question. Seizures originating from the supplementary motor zone (SMZ) are characterised by stable clinical manifestations: they begin in childhood and persist unchanged in adults [1].

Frontal hyperkinetic seizures

Some seizures originating in the frontal lobes of the brain exhibit marked motor symptoms in the absence of tonic limb placement. Such paroxysms have been described as frontal seizures with abnormal behaviour, frontal hypermotor seizures, frontal complex partial seizures, and frontal hyperkinetic seizures (the latter is the preferred term). Characteristic hyperkinetic automatisms are apparently a highly specific feature of this type of frontal seizure. At the same time, there are descriptions of nocturnal hyperkinetic paroxysms originating from the temporal lobe. Apparently, the frontal system responsible for such ictal manifestations can be activated by irritation of various brain regions [4]. Despite their flamboyance and bizarre nature reminiscent of hysterical seizures, HPs are characterised by stereotypical ictal manifestations in each clinical case. As with other types of frontal seizures, there are "transitional" forms between GPs and PDMS, GPs and medial temporal seizures (in which case the paroxysms usually begin as GPs and then progress to temporal seizures), GPs and seizures originating from the sensorimotor cortex (the latter, however, are usually not part of the GP). GPs can occur with foci in different parts of the frontal lobe, mainly in the orbitofrontal and frontal polar regions, the anterior cingulate or the medial parts of the frontal lobe [5].

The interictal EEG shows slowing of the theta and delta ranges and epileptiform discharges in frontal leads. During a seizure, EEG showed flattening of the curve or low beta activity, disappearance of a pronounced interictal EEG abnormality, or the appearance of theta rhythm [5].

Like PDMS, GPs can be characterised by a normal or non-specific pattern of interictal and, in many cases, ictal EEG, which leads (along with flamboyant motor manifestations) to misdiagnosis of non-epileptic seizures. This makes it necessary to have a detailed knowledge of the semiotics of seizures - the main criterion for differential diagnosis of HP and psychogenic paroxysms. The latter are more characterised by rhythmic pelvic movements, left-right head turns, striking movements, and in many cases there is an aggravated history of mental illness. Short, clustered, often nocturnal attacks (usually beginning at a young age) and pronator positioning of the limbs at the time of the attack are more typical of GP.

Diagnosis of HP, like PDMS, is based on a set of features, which, in addition to those described above, also include usually normal EEG and MRI data. It should be borne in mind that all the clinical and instrumental findings characteristic of PD are fully consistent with another frontal lobe epileptic syndrome with a genetic defect, autosomal dominant nocturnal frontal lobe epilepsy.

Absences

Absences in some cases occur in discharges with a predominant localisation in the frontal lobe of the brain, mainly in the orbitofrontal and medial frontal regions [6]. They may not differ clinically from similar seizures with diffuse spread of epiactivity. Opercular seizures beginning in the eponymous formation that is part of the frontal lobe are described less frequently than other types of frontal seizures. Clinical manifestations of such paroxysms include profuse salivation, orofacial apraxia with possible clonic convulsions in the muscles of the face, neck, and larynx, tingling sensation in the throat and half of the face or contralateral upper extremity [7].

Paroxysms originating from the temporal lobes of the brain

In a proportion of patients, frontal seizures may resemble paroxysms emanating from the temporal lobes of the brain. As described above, GPs originating in the orbitofrontal cortical region may spread to medial temporal structures, leading to the transformation of dramatic ictal manifestations of GPs into milder ones characteristic of medial temporal lobe epilepsy (MTE). In other cases, orbitofrontal seizures are initially clinically and electroencephalographically indistinguishable from temporal seizures, which, in the absence of focal pathology on MRI, often leads to errors in diagnosis [8].

Temporal lobe epilepsy

Temporal lobe epilepsy (TLE) accounts for more than half of all cases of focal epilepsy in adults. Although the development of VE was classically previously attributed to isolated damage to temporal structures such as the hippocampus and amygdala, more recent studies have shown widespread cortical atrophy involving both temporal and extratemporal regions [9].

Symptomatic and cryptogenic VE can be divided into 2 types according to well-defined syndromes: 1) medial, or amygdalo-hippocampal, associated with the lesion of the limbic system in the deep parts of the temporal lobe; 2) lateral, or neocortical, based on the lesion of the temporal neocortex.

Medial temporal lobe epilepsy

Hippocampal sclerosis (HS) is the most common pathological substrate for medial temporal lobe epilepsy (MTLE) with a characteristic signature of death of major neurons, primarily in CA1 and the hilus (the dentate gyrus layer of the hippocampus). Other cytoarchitectonic abnormalities (in particular, dispersions of granular cells of the dentate gyrus and abnormalities of the hilus cytoskeleton) have been identified in other areas of the hippocampus [10].

Patients often have a history of childhood febrile seizures, as well as central nervous system trauma and infections [11]. Temporal seizures are usually preceded by an aura that includes viscerosensory sensations (especially often in the epigastric region), fear, anxiety, and drowsiness. The aura is usually followed by stiffening, staring at a single point (starring), and pupil dilation. If the seizures end there, they may be confused with absences (temporal absences). Oroalimentary (chewing, tweezing and licking the lips) and motor automatisms (gesturing, groping and picking up objects) are possible.

Age evolution of the attacks is noted until the age of 10 years, when the clinical picture becomes indistinguishable from that of VE in adults. In children up to 3 years of age, tonic and clonic motor manifestations predominate, and then the seizures usually become hypokinetic, with the incidence of automotor seizures steadily increasing with age [12].

The postictal phase in VTE with HS is characterised by prolonged confusion, disorientation and speech disturbances, longer when the ictal focus is localised in the dominant hemisphere, which distinguishes this syndrome from extratemporal seizures, for which postictal confusion is less characteristic.

Routine EEG findings are often normal or nonspecific, but with prolonged EEG monitoring, epileptiform activity characterised by peaks, sharp and slow waves, and their combination, is detected in the anterior temporal leads in the majority of cases, often bilaterally independent [13].

Medial temporal lobe epilepsy

Hippocampal sclerosis (HS) is the most common pathological substrate for medial temporal lobe epilepsy (MTLE) with a characteristic signature of death of major neurons, primarily in CA1 and the hilus (the dentate gyrus layer of the hippocampus). Other cytoarchitectonic abnormalities (in particular, dispersions of granular cells of the dentate gyrus and abnormalities of the hilus cytoskeleton) have been identified in other areas of the hippocampus [10].

Patients often have a history of childhood febrile seizures, as well as central nervous system trauma and infections [11]. Temporal seizures are usually preceded by an aura that includes viscerosensory sensations (especially often in the epigastric region), fear, anxiety, and drowsiness. The aura is usually followed by stiffening, staring at a single point (starring), and pupil dilation. If the seizures end there, they may be confused with absences (temporal absences). Oroalimentary (chewing, tweezing and licking the lips) and motor automatisms (gesturing, groping and picking up objects) are possible.

Age evolution of the attacks is noted until the age of 10 years, when the clinical picture becomes indistinguishable from that of VE in adults. In children up to 3 years of age, tonic and clonic motor manifestations predominate, and then the seizures usually become hypokinetic, with the incidence of automotor seizures steadily increasing with age [12].

The postictal phase in VTE with HS is characterised by prolonged confusion, disorientation and speech disturbances, longer when the ictal focus is localised in the dominant hemisphere, which distinguishes this syndrome from extratemporal seizures, for which postictal confusion is less characteristic.

Routine EEG findings are often normal or nonspecific, but with prolonged EEG monitoring, epileptiform activity characterised by peaks, sharp and slow waves, and their combination, is detected in the anterior temporal leads in the majority of cases, often bilaterally independent [13].

The clinical features of VLE include the absence of febrile seizures in the history, the presence of experimental (experience-based) auras (auditory and visual illusions and hallucinations), contralateral dystonic attitude and loss of contact from the first seconds of the seizure, lower frequency of motor manifestations and absence of oroalimentary automatisms, short duration of seizures, and greater propensity for secondary generalisation compared with PHE. The clinical picture of seizures in VLE is not fundamentally different from autosomal dominant VLE (familial VE with auditory manifestations) [20].

The long-term prognosis in VLE does not differ from that of other forms of symptomatic focal epilepsy; surgical intervention is indicated in patients if antiepileptic drugs are ineffective.

Parietal epilepsy

In contrast to the repeatedly described temporal and frontal lobe epilepsies, seizures originating from the parietal lobes of the brain have been described in only a few publications. This is due to the relatively lower prevalence and difficulties in diagnosing parietal epilepsy (PE), which is due, in turn, to the anatomo-physiological features of the parietal lobes of the brain.

As already noted, some clinical manifestations of seizures beginning in the parietal lobe of the brain have been known since antiquity, but patients with such ictal manifestations are infrequently encountered in practice [21].

In the first decade of life, TE in the overwhelming number of cases is a consequence of organic brain damage (meningoencephalitis, severe craniocerebral trauma, perinatal lesions). In the practice of an "adult" epileptologist, patients with cryptogenic forms, which debut without apparent causes in the 2-3rd decade of life, are most often encountered. The frequency of seizures may vary from single seizures over the entire period of the disease to daily episodes. Classical symptoms of TE are represented by paresthesias, usually contralateral to the seizure focus. Patients describe ictal somatosensory symptoms as "goosebumps crawling," "numbness," "tingling," and "twitching." Examples of frequent pain sensations during a TE attack include diffuse headache, diffuse abdominal pain accompanied by vomiting and diarrhoea, lateralised pain sensations in the trunk and extremities ("electric shocks").

Seizures originating from the anterior inferior parietal lobe of the dominant hemisphere may manifest with speech disorders. Ictal activity restricted to the parietal lobe leads to the development of apraxia and agnosia, and the patient appears confused and disorientated during the seizure, which, however, is common in virtually all partial seizures.

Quite often patients have ictal vertigo ("epileptic tornado") associated with irritation of the central section of the vestibular analyser located in cortical field 2v of the parietal lobe, occupying the base of the intraparietal sulcus, immediately behind the area of hand and mouth representation in the postcentral gyrus. If ictal activity spreads to the temporal lobes, clinical manifestations may include auditory hallucinations, automotor seizures, or dialeptic temporal absences. If seizure activity involves the cortex at the junction of the temporal, parietal and occipital lobes of the brain, it may be manifested by complex visual and auditory hallucinations accompanied by altered consciousness (dreamy state).

Occipital epilepsy

Occipital epilepsy (OE) has received relatively little attention in the literature, as have parietal epilepsies.

The prevalence of ZE is less than 10% of all focal epileptic syndromes. The frequency of seizures varies from single seizures over the entire course of the disease to daily seizures. Ictal visual symptoms can be either positive in the form of "flashes", "circles", "goosebumps", "birds", "flickering bright spots", etc., or negative in the form of "darkness before the eyes" or "black spots" (black scotomas) [22]. Various oculomotor symptoms are found in occipital seizures, such as tonic retraction of the eyeballs. Autonomic manifestations in the form of nausea and/or vomiting at the time of the seizure have been reported [23]. The uncommon combination of ZE with headache gave rise to the term "migraine-epilepsy", still found in domestic medical records.

Conclusions: Knowledge of the semiotics of epileptic seizures arising as a result of abnormalities in various brain lobes is of significant clinical importance primarily for the detection of epileptogenic structural brain lesions and/or as part of preoperative examination in patients with intractable epilepsy [24].

Literature:

1. Рудакова И.Г., Котов С.В., Котов А.С. Возрастные аспекты эпилепсии у взрослых. Нервные болезни. 2007;4:2–9.
2. Kellinghaus C., Lüders H.O. Frontal lobe epilepsy. Epileptic Disord. 2004;6(4):223–239.

3. Kellinghaus C., Kotagal P. Lateralizing value of Todd's palsy in patients with epilepsy. Neurology. 2004;62:289–291.
4. Nobili L., Cossu M., Mai R. et al. Sleep-related hyperkinetic seizures of temporal lobe origin. Neurology. 2004;62(3):482–485.
5. Staack A.M., Bilic S., Wendling A.S. et al. Hyperkinetic seizures in patients with temporal lobe epilepsy: clinical features and outcome after temporal lobe resection. Epilepsia. 2011;52(8):1439–1446. DOI: 10.1111/j.1528-1167.2011.03100.x.
6. Niedermeyer E. Epileptic seizure disorders. In: Niedermeyer E., Lopes da Silva.F., eds. Electroencephalography: basic principles, clinical applications, and related fields. Baltimore: Urban and Schwarzenberg, 2000
7. Р.Дж. Халимов, А.М. Джураев Критерии оценки мультиспирально–компьютерно–томографического исследования детей с болезнью Пертеса. Материалы-III съезда травматологов–ортопедов Республики Казахстан и VII Евразийского конгресса травматологов-ортопедов. 2019/10. С.3-4
8. Smith J.R., Sillay K., Winkler P. et al. Orbitofrontal epilepsy: electroclinical analysis of surgical cases and literature review. Stereotact Funct Neurosurg. 2004;82(1):20–25
9. Mueller S.G., Laxer K.D., Cashdollar N. et al. Voxel-based optimized morphometry (VBM) of gray and white matter in temporal lobe epilepsy (TLE) with and without mesial temporal sclerosis. Epilepsia. 2006;47(5):900–907.
10. Thom M., Sisodiya S.M., Beckett A. et al. Cytoarchitectural abnormalities in hippocampal sclerosis. J Neuropathol Exp Neurol. 2002;61(6):510–519.
11. Patterson K.P., Baram T.Z., Shinnar S. Origins of temporal lobe epilepsy: febrile seizures and febrile status epilepticus. Neurotherapeutics. 2014;11(2):242–250. DOI: 10.1007/s13311-014-0263-4.
12. Ray A., Kotagal P. Temporal lobe epilepsy in children: overview of clinical semiology. Epileptic Disord. 2005;7:299–307.
13. Javidan M. Electroencephalography in mesial temporal lobe epilepsy: a review. Epilepsy Res Treat. 2012;2012:637430. DOI: 10.1155/2012/637430.
14. Engel J Jr. Functional explorations of the human epileptic brain and their therapeutic implications. Electroencephalogr Clin Neurophysiol. 1990;76:296–316.
15. Guedj E., Bonini F., Gavaret M. et al. 18FDG-PET in different subtypes of temporal lobe epilepsy: SEEG validation and predictive value. Epilepsia. 2015;56(3):414–421. DOI: 10.1111/epi.12917.
16. Kumar A., Chugani H.T. The role of radionuclide imaging in epilepsy, Part 1: Sporadic temporal and extratemporal lobe epilepsy. J Nucl Med. 2013;54(10):1775–1781. DOI:
17. Котов А.С., Руденко А.М. Криптогенные и симптоматические височные эпилепсии у взрослых. Анналы клинической и экспериментальной неврологии. 2009;3(2):9–13.
18. Wiebe S., Blume W.T., Girvin J.P. et al. A randomized, controlled trial of surgery for temporal lobe epilepsy. N Engl J Med. 2001;345:311–318
19. Chabardes S., Kahane P., Minotti L. et al. The temporopolar cortex plays a pivotal role in temporal lobe seizures. Brain. 2005;128:1818–1831
20. Michelucci R., Poza J.J., Sofia V. et al. Autosomal dominant lateral temporal epilepsy: clinicalspectrum, new epitempin mutations, and genetic heterogeneity in seven European families. Epilepsia. 2003;44(10):1289–1297.
21. Ikeda A., Matsumoto R., Ohara S. et al. Asymmetric tonic seizures with bilateral parietal lesions resembling frontal lobe epilepsy. Epileptic Disord. 2001;3(1):17–22.
22. Котов А.С., Рудакова И.Г., Белова Ю.А., Котов С.В. Затылочная эпилепсия у взрослых. Журнал неврологии и психиатрии им. С.С. Корсакова. 2009;109(7):4–8.
23. Adcock J.E., Panayiotopoulos C.P. Occipital lobe seizures and epilepsies. J Clin Neurophysiol. 2012;29(5):397–407.
24. Куташов В.А., Куташова Л.А. Изменение психического статуса у больных эпилепсией. РМЖ. 2014;16:1153.
25. Котов А.С. Оперативные вмешательства и обезболивание у больных эпилепсией. Клиническая лекция. РМЖ. 2015;12:710.

ISSN 2181-0982

Doi Journal 10.26739/2181-0982

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

ТОМ 4, НОМЕР 5

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH

VOLUME 4, ISSUE 5

Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC the city of Tashkent,
Amir Temur Street pr.1, House 2.
Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

Контакт редакций журналов. www.tadqiqot.uz
ООО Tadqiqot город Ташкент,
улица Амира Темура пр.1, дом-2.
Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000