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NEW APPROACHES TO THE TREATMENT OF MENTAL DISORDERS IN PATIENTS WITH EPILEPSY

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Abstract
Today, the treatment of mental disorders that develop as a result of epilepsy is one of the Doric problems of psychiatry and neurology. The prevalence of mental disorders in epilepsy is becoming one of the pressing issues of modern medicine.

Key words
Epilepsy, treatment, complication, mental disorder.

Introduction
The risk of death in people with uncontrolled epilepsy significantly exceeds the figure in the general population [1]. According to various researchers, the risk level ranges from 1.4 to 3.6 [2-4]. The main causes of death are an epileptic condition and its complications, injuries and sudden death in epilepsy (sudepen - unexpected death in epilepsy). Early full onset of treatment prevents the development of pathological personal changes and improves the quality of life of patients [5]. Unfortunately, against the background of conservative treatment, 30% of patients cannot fully control seizures and develop pharmacoresistance. The latter concept refers to the inability to control seizures when using two or more adequate schemes for the use of anticonvulsant drugs as monotherapy or a combination of them [6]. Each such patient should be seen as a candidate for surgical treatment for epilepsy.

The effectiveness and safety of surgical treatment of epilepsy has been proven in many studies [7-13]. Thus, for example, in the surgical treatment of magnetic resonance (Mr)-positive transient forms of epilepsy, complete loss of seizures can be achieved in more than 80% of patients. At the same time, in non-temporal forms of epilepsy without structural pathology (Mr-negativity) in magnetic resonance tomograms (MRI), the results of surgical treatment turn out to be worse, and according to various authors, only 40-50% of patients achieve release from seizures [1, 14]. This is due to relatively simple localization of the epileptogenic zone in mrpositive temporal forms of epilepsy and complex localization of focus in Extratemporal Mrnegative forms of the disease.

Epilepsy is one of the oldest known neurological disorders in the world and is characterized by a wide range of neuropsychic disorders in the clinical picture. Neuropsychiatric disorders in epilepsy are one of the important components of the clinical picture of the disease and have a significant impact on the quality of life and social adaptation of patients. These include cognitive, affective and behavioral disorders, among which the leading place belongs to disorders in the cognitive sphere [9-13]. There is increasing evidence that epilepsy may be associated with joint conditions such as learning problems, psychological and behavioral disorders. Comorbid states differ in type and severity, ranging from subtle learning difficulties to specific disorders of intellectual and mental function [14]. When diagnosing a patient with epilepsy, it is important to pay early attention to the presence of comorbid conditions to ensure their early identification, diagnosis and appropriate control [15]. Cognitive impairment in epilepsy patients defined by a neuropsychological test is

VOLUME 4, ISSUE 2, 2023

determined by complex interactions of biological and social factors, in some cases variable, and may have some differences depending on selected research methods [16].

The scientific community has formed general ideas about the change in cognitive functions in epilepsy, which include: a decrease in short-term memory, an increase in information units and a decrease in attention to their reproduction, subsequent memory and memorization disorders, inertia of mental processes, the definition of thinking as rigid and sticky; characteristic speech changes-slowing down, oligophasia; changes in the intellectual-Mnestic and emotional-personal sphere [17].

Changes in cognitive functions in the structure of epileptic disease were found in antiquity and the ancient Eastern periods [18]. A systematic approach to the analysis of scientific data can be observed in the works of the French psychiatrist Esquirol [19], who compiled tables of clinical signs of epilepsy and recorded dementia in patients, as well as post-mortem memory disorders. He noted that the development of mental disorders increases the development of the disease and causes dysfunctional dementia. publishes an epidemiological study, noting for the first time post-mortem changes in the hippocampus in patients with epilepsy. Later Sommer W. [20] studied the hippocampal region at autopsy and found selective loss of gliosis and pyramidal neurons in the hippocampal CA1 area, commonly known as hippocampal sclerosis. These changes were combined with the presence of dementia. The first psychodiagnostic studies were carried out in the late 19th - early 20th centuries. and V. M. Bekhterev, E. Crepelin, A. N. Related to the Menas of Bernstein and other scholars [21]. V. M. Bekhterev studied the development of short-term amnesia in patients with epileptic seizures and demonstrated that memory impairment may have epileptic characteristics [22]. L. S. Vygotsky emphasized the importance and need for a comprehensive diagnosis of epilepsy in children, taking into account violations of gni functions for timely correction [23]. V. Penfield and B. Milner [24-30] figured out the role of epileptic focus and played a large role in neuropsychological diagnosis of the state of higher mental functions.

Kn and behavioral disorders can appear even before the debut of epilepsy, appear after the onset of seizures and develop with the development of the disease [31]. Among the most common manifestations of cognitive dysfunction in epilepsy are depression, memory, attention, and bradyphrenia disorders [32-35]. Various factors play an important role in the pathogenesis of the aforementioned disorders: organic damage to brain structures, neuronal dysfunction, interstitial epileptic activity, recurrent seizures, as well as taking certain antiepileptic drugs (AEP) [36-38].

It should be noted that one patient may have several indicated causes at the heart of cognitive and mental disorders (as a rule, this is the case). It is very difficult to assess the contribution of certain factors, since different factors can independently have different effects on cognitive disorders in epilepsy. Currently, many studies are underway to assess the independent contribution of various factors to the development of cognitive and mental disorders in epilepsy [39].

One of the factors that negatively affect cognitive function is the etiology of epilepsy. The causative factors can be acquired diseases (traumatic brain injury, stroke, neuroinfection consequences, hypoxic-ischemic perinatal brain injury, mesial temporal sclerosis, etc.) and genetic diseases (tuberous sclerosis, Fragile X syndrome, retta, Dravet, etc. More recently (2017), the term" developmental encephalopathy " (developmental Encephalopathy) has been introduced, meaning that cognitive and behavioral disorders in developmental encephalopathies are caused by the disease itself (e.g. genetic syndrome) and their course does not depend on the treatment of AEP [40].

Although the etiology of epilepsy plays a leading role for cognitive development, seizures with debut at an early age, regardless of the etiology of the disease, can lead to cognitive impairment [41]. Thus,

VOLUME 4, ISSUE 2, 2023

in the study of neuropsychological functions in children with resistant epilepsy caused by focal cortical dysplasia, Corman B. etc.

it has been shown that the main factors associated with kn are the debut age of epilepsy and the prevalence of dysplasia (its localization played a lesser role), each of these factors contributing independently to the formation of kn [42]. D. S. O'leary et al. [43] the disease recorded more pronounced abnormalities in the mental field in children under 5 years of age, and further development of changes was associated with the polyformity of seizures and the appearance of new types of them during the disease. These observations can be compared to work data [44], which lists the research results of a group of adult patients. A significant difference in the cognitive field was noted in patients with epilepsy under 5 years of age and later onset. When the disease occurs at the age of 14-15 years and after the age of 18 years, the association with kn has not been determined [45, 46].

In many studies, the duration of the disease [47, 49] was not a specific factor in the development of memory disorders. A number of authors have shown that temporal epilepsy [48] has a relationship between the duration of the disease and kn. In some cases, in Primary general epilepsy, kn is associated with the duration of the disease, but correlation is observed in children under 11 years of age [50].

Many researchers associate the deterioration of cognitive ability with the frequency of seizures [51, 52]. In monozygotic twins with identical forms of epilepsy, but frequent seizures, kn development was found at different frequency of seizures [53].

According to these materials, there is a clear relationship between the localization of kn and paroxysmal focus.

In particular, in temporal epilepsy, the cognitive sphere is most tormented by the fact that the focus is located in the mediobasal parts of the temporal lobe. In the left-sided temporal focus, verbal memory is impaired, while on the right, spatial and visual memory is impaired [54, 55]. With the localization of focus in the left hemisphere, auditory speech disorders and long-term memory are characteristic, and in the right hemisphere, visual memory disorders, the severity of verbal and spatial disorders [56, 57, 59]. At the same time, the weakening of these functions depends on the dominance of the hemispheres.

Kn is closely related to the phenomenon of brain aging. Therefore, this problem is associated with cognitive function and is of great importance in elderly and elderly patients. The main causes of the development of epilepsy in this category of people are premorbid Insolvency and cerebrovascular pathology, stroke, trauma and neurodegenerative diseases [58, 60].

Epilepsy is a disease that requires long, many years (at least 3 years after the end of the seizure) of therapy. According to the latest recommendations of the international anti-epileptic League (anti-epileptic internationio nal League, ILAE), epilepsy can be considered permitted (resolved) if the patient has not had seizures for 10 years, but also if the patient has advanced age. age-related forms of epilepsy [61].

Currently, it has been shown that almost all AEPS can affect cognitive functions, the affective sphere, and the behavior of patients, but this effect can be both positive and negative [62]. In 1926, L. Muskens [63] argued that personality changes may have been the result of the use of such therapy, specifically barbiturates. According to research, 30-60% of patients view memory and thought slowing problems as side effects of AEP, but many studies have shown that cognitive impairments were present even before treatment began [64]. There is debate about the differences between the

VOLUME 4, ISSUE 2, 2023

side effects of old and new AEPS. In the treatment of epilepsy, monotherapy is preferred, in which such effects are less noted [65]. However, even the use of one drug can cause kn. There is evidence of the same decrease in cognitive function in patients taking carbamazepine, phenytoin, and Phenobarbital [67].

Purpose of the study: optimization of therapy for patients suffering from pharmacoresistant forms of epilepsy with mental disorders in conditions of multidisciplinary interaction.

Materials and methods Using the clinical-psychopathological method, a comprehensive mental state assessment scale, a general clinical impression scale, a side effects scale and an eclipse Assessment Scale, 42 patients were examined (men-29 (69%), women - 13 (31%) people), with an average age of 42,1 ± 1,64 years, in dispensary observation at HDPE, diagnosed with epilepsy, pharmacoresist form. 9 (21,4%) of patients have a focal symptomatic form, 21(50%) a focal cryptogenic Form, 3 (7,1%) a general idiopathic Form, 8 (19,1%) a general cryptogenic, 1 (2,4%) A general symptomatic form. 34 (80.9%) people have been diagnosed with epilepsy-induced personality disorder (F 07.02), 8 (19,1%) with epilepsy-induced dementia (F02.802). Among concomitant mental disorders, the following were also noted: 31(73,8%) patients had anxiety, 14 (33,3%) patients had depression, 25 (59,5%) patients had dysphoria, all 42 patients had different levels of cognitive impairment (100%), so 17 (40,4%) people received co-therapy: risperidone 8 (19,1%) people, aminazine 9 (21,4%) people, fluvoxamine 8 (19,1%) people, nootropics 23 (54,7) people. Metabolic disorders (overweight) were reported in 5 (11,9%) patients and insomnia in 2 (4,7%) patients. The duration of the disease is 28.5 ± 3,9 years. Total seizures of up to 1-2 times a day were observed in all 42 patients. All patients had a disability group, none of which worked. For all patients, an integrated approach was implemented in the diagnosis and treatment of patients by the method of interaction of brigades. The Brigade consisted of: doctor-psychiatrist-Brigade leader, neurologist, neurosurgeon, psychotherapist, psychologist, social worker. The complex interaction program is developed separately for each patient.

Results and discussions Under an individual plan, patients underwent further examination using EEG video monitoring, MRI, and PET techniques. Given the duration of therapy and resistance to seizures, the psychiatrist, together with the neurologist, replaced the current therapy with the removal of one of the drugs in polyterapy or the addition of a second drug to monotherapy and the transition to therapy with the drug lacosamide ("vimpat")., by titration of the dose for 1 month, up to a therapeutic dose of 300-400 mg per day. Group training with 36 (85,7%) patients was carried out by the psychologist in order to socialize patients, their intelligence was maintained (slightly decreased), frequency 2 times a week, aimed at increasing the Coordination of therapy, expanding methods of social interaction. The psychotherapist conducted psycho-educational activities with patients and their relatives. The social worker provided assistance in the interaction of patients with administrative and social security structures. After 6 months of observation, the following results were obtained: the absence of seizures in 13 (30,9%) people, a 50% decrease in baseline rates in 19 (45,2%) patients, an increase in seizures in 2 patients with a common cryptogenic form (4,7%), in 8 (19,1%) patients. the status of the attacks remained unchanged. 29 (69%) neurosurgical consultations were performed on patients with low remission (non-remission) seizures, and 6 (14,3%) patients were offered to install a VNS stimulator. At a dose of 400 mg per day in 6 patients. dizziness and headaches were noted, so the dose of the drug was reduced to 300 mg per day., 1 patient showed increased drowsiness in the combination of vimpate with benzodiazepines, the dose of the drug was reduced to 200 mg per day. No changes in the cardiovascular, circulatory system have been recorded.

VOLUME 4, ISSUE 2, 2023

With the decrease in attacks, the mental state of patients improved, with 5 (11,9%) patients managing to gradually abolish fluvoxamine, while 10 (23,8%) patients had their antipsychotics abolished. The overall score on the all-round mental assessment scale improved to an average of 3,2±0,6 points, and on the CGI scale to 2,1±0,3 points. 31 (73,8%) patients recorded an improvement in interaction within the family, a decrease in the manifestation of aggression, a decrease in internal conflicts, 4 (9,4%) of the patient's employment.

Conclusion This, during the study, it was found that multidisciplinary interaction of specialists in the field of psychoneurology is necessary to optimize therapeutic and rehabilitation measures for patients with epilepsy with mental disorders. To date, scientists ' opinions on the nature and pathogenesis of cognitive dysfunction in epilepsy have not been unanimously consensus. Due to the heterogeneity of the etiology of the development of the disease, the influence of the external environment and active intervention in the process of therapeutic measures, each factor can have its own significance, depending on the context. Based on the above, it should be noted that in modern Epileptology the problem of kn is not solved in any way.

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VOLUME 4, ISSUE 2, 2023

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