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LATE CLINICAL AND NEUROIMAGING MANIFESTATIONS OF POST-TRAUMATIC EPILEPSY AND OPTIMIZATION OF ITS TREATMENT

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MONOGRAPH

Compiled by Zokirov Muzaffar, Muhammadjonov Oqilbek

India-2023

This monograph is devoted to one of the most topical topics of modern neurology, such as epilepsy. Post-traumatic epilepsy is the most common form of post-traumatic epilepsy, and with the development of urbanization, its incidence is increasing.

In this work, a scientific analysis of patients with distant post-traumatic epilepsy is carried out, in which the first convulsions occur after 3 years from the moment of brain injury. Electroencephalogram, magnetic resonance imaging, studies of neurocognitive functions were studied in patients, and nootropic drugs were used to correct them.

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INTRODACTION

Epilepsy is a widespread disease with an overall incidence of 1-2%, ranking third among brain diseases. More than 50 million people suffer from this disease. Every year, 50-70 thousand cases of this disease are registered per 100,000 people. From thousand patients the disease in 1-5 cases ends in death. (Internet Journal of Neurosurgery CNS 2007).

According to the classification, all forms of epilepsy are divided by etiology into symptomatic, cryptogenic and idiopathic. Symptomatic epilepsy is a polyetiological disease. Causes of symptomatic epilepsy can be volumetric formations, neuroinfections, vascular diseases, injuries). The leading etiological factor in symptomatic epilepsy of young age is traumatic brain injury (TBI), which occupies 30-50% of all types of injuries in peacetime (Khilko V.A. et al., 1989; Guzeva V.I., 1992, 2007 ; Gromov S.A., Lobzin BC, 1993; Gaidar B.V. et al., 1996; Parfenov V.E., Svistov D.V., 2002; Odinak M.M. et al., 2003, 2004). The incidence of post-traumatic epilepsy in cases of previous TBI is, according to numerous studies, from 5 to 50%. In symptomatic forms of epilepsy, the morphological substrate of the disease is verified according to neuroimaging data.

In recent years, the number of publications devoted to the study of cognitive functions in various neurological diseases has increased [6, 62, 114]. The problem of increasing cognitive deficit is considered in close connection with the progression of the pathological process, including in symptomatic forms of epilepsy in adults [70, 72, 55, 106].

Today, cognitive impairment is no longer an absolutely incurable condition. In most cases, modern methods of symptomatic and pathogenetic therapy can achieve clinical improvement or temporary stabilization of the condition and improve the quality of life of the patient and his relatives [1, 3, 43, 80, 89]. At the same time, timely diagnosis and the earliest possible start of therapy for cognitive disorders are important. Currently, unfortunately, there is a

serious problem of late diagnosis of cognitive impairment, often only at the stage of severe dementia, when the therapeutic options are already small.

Early identification of individuals who may subsequently develop dementia is one of the most relevant areas of research in epileptology. The importance of the study of cognitive disorders is obvious and due to the fact that the timely detection of these disorders undoubtedly improves the quality of life of patients and expands the possibilities of therapeutic intervention, which, ideally, can delay or even prevent the onset of social maladaptation.

An analysis of the literature showed that in recent decades, issues related to clinical symptoms, pathogenesis, development predictors and risk factors, and the diagnosis of post-traumatic epilepsy have been studied [12, 13, 62, 87, 124, 105]. Works describing the pathology of higher nervous activity in epilepsy primarily concern gross personal and intellectual-mnestic disorders [7, 81, 110]. At the same time, the issues of assessing cognitive functions, their timely medical correction in post-traumatic epilepsy are not sufficiently covered in the literature and are controversial.

Purpose of the study :

To study distant clinical and neuroimaging manifestations of post-traumatic epilepsy and develop an optimization of its treatment

Research objectives:

1. To study the long-term clinical manifestations of post-traumatic epilepsy depending on the length of time and the course of the disease
2. To study neuroimaging manifestations of post-traumatic epilepsy, to compare clinical and neurophysiological data
3. To develop and substantiate the expediency of complex differentiated therapy for post-traumatic epilepsy.

Materials and methods of research:

To achieve this goal, it was surveyed from 2020 to 2023. 42 patients suffering from symptomatic post-traumatic epilepsy with a limitation period of 3

years or more. The studies were carried out in the departments of emergency neurology, neurology, neurosurgery and the consultative polyclinic of the clinics of the Ferghana Medical Institute of Public Health.

1. Clinic - neurological studies.

2. Paraclinical research methods

- MRI, CT scan of the brain.
- Electroencephalography.
- Neuropsychic Research Methods

A) Mental status assessment scale, MMSE (Folstein M. _ F. _ et al , 1975)

B) Battery of tests for assessing frontal dysfunction - BTLD (English: Firontal Assessment Battery - FAB)

a- Khanin test to detect anxiety

3. Statistical analysis.

Scientific novelty

The features of the course of post-traumatic epilepsy in patients with an experience of 3 years or more, the state of the cognitive and psycho-emotional state according to clinical, neuropsychological, electrophysiological examinations are described.

The dependence of the degree of cognitive deficit on the state of the bioelectrical activity of the cerebral cortex is shown. The effectiveness of complex drug therapy with antiepileptic and neuroprotective drugs was studied.

Structure and scope of the monograph:

The dissertation is presented on 116 pages of computer text. Illustrations: 3 figures, 28 tables. It consists of an introduction, a literature review, research methods, 1 chapters of own research, a conclusion, conclusions, practical recommendations and a literature index containing 57 Uzbek-Russian, 60 foreign sources.

Conclusions

1. Post-traumatic epilepsy is dominated by moderate and severe cognitive impairment with a predominant impairment of memory and concentration. As

the disease progresses, the apracto-agnostic syndrome joins, a violation of the understanding of complex logical and grammatical structures.

2. Psycho-emotional status in patients with post-traumatic epilepsy is characterized by a decrease in reactive anxiety and an increase in personal anxiety as the disease progresses.

3. The use of Phenotropil has a positive effect on the cognitive sphere of patients, regresses the development of reactive anxiety disorders, leads to a decrease in epileptic activity and normalization of the neurophysiological state

Practical recommendations

1. For a more reliable diagnosis of the degree of cognitive impairment in and post-traumatic epilepsy, it is advisable to use several complementary methods.

2. In the complex therapy of cognitive disorders in patients with epilepsy with a predominance of reactive anxiety disorders, and with impaired attention and memory, Phenotropil should be used.

LIST OF ABBREVIATIONS :

AEP - antiepileptic drug

ICH - intracranial hypertension

PCA - posterior cerebral artery

CD - cognitive disorders

CT - computed tomography

LSC - linear velocity of blood flow

ICD - international classification of diseases

MRI - magnetic resonance imaging

ACA - acute cerebrovascular accident

PI - pulsation index

ACA - anterior cerebral artery

PTE - post-traumatic epilepsy

PE - post-traumatic encephalopathy

MCA - middle cerebral artery

CSF - cerebrospinal fluid

CAS - condition after surgery

MCI - moderate cognitive impairment

CNS - central nervous system

TBI - traumatic brain injury

EEG - electroencephalography

FAB - - frontal battery assessment

ILAE - International League Against Epilepsy

IQ - Intellectual Quality

MMSE - Mini Mental State Examination

CONTENT

Introduction	**3 page**
List of conventional abbreviations	**7 page**
Chapter I. _ Literature review	
Epidemiology	10 page
Traumatic seizures and post-traumatic epilepsy	11 page
Pathogenesis of post-traumatic epilepsy	13 page
Classification of epileptic seizures.	20 page
Clinical picture of post-traumatic epilepsy	26 page
Diagnosis of post-traumatic epilepsy	32 page
Treatment of post-traumatic epilepsy	36 page
Chapter II. Materials and methods of research	
Characteristics of the examined patients	42 page
Research methods	45 page
Radiation and neurophysiological diagnostics	47 page
Statistical processing of results	48 page
Chapter III. **Research results**	
Clinical and neurological symptoms	49 page
The state of the general mental status of the examined	52 page
Features of sensorimotor reactions and attention	54 page
Features of cognitive disorders in patients with post-traumatic epilepsy depending on the antiepileptic drug taken	68 page
Functional state of the cerebral cortex according to electroencephalography data	72 page
Dynamics of indicators of the neuroimaging research method.	
Dynamics of cognitive disorders against the background of complex therapy	76 page
Conclusion	**86 page**

Conclusions **91 page**

Practical recommendations **92 page**

References **93 page**

Applications **103 page**

CHAPTER 1. MODERN PROBLEMS OF POSTTRAUMATIC EPILEPSY

1.1. Epidemiology

The prevalence of traumatic brain injury in the world is 4 cases per 1000 population. One of the consequences of brain injury is post-traumatic epilepsy (PTE), which develops in 2-53% of TBI survivors. The frequency and rhythm of epileptic seizures in post-traumatic epilepsy largely depend on the severity of TBI, the location and volume of the focus of brain damage, and the state of the premorbid background. The most relevant aspects of epilepsy are social and psychological problems, which have become especially significant in connection with the democratization of our society and the increasing level of the role of a person in it. Society is becoming more and more interested in making its labor potential higher and, therefore, directs great efforts to prevent disability, due to the presence of various paroxysmal disorders in some of its members, including epilepsy. Obtaining control over seizures is not only of great clinical importance, but also indirectly affects the social status of patients, their quality of life, since a seizure is the main destabilizing factor (Konovalov A.N. et al. 1998; Yakhno N.N., 2007; Gusev E.I., Konovalov A.N., 2010; Chang BS, Lowenstein D.H., 2003; Agrawal, 2006; Pitkanen, 2007).

A post-traumatic epileptic seizure (epileptic syndrome) is usually a single epileptic paroxysm that occurs after a head injury in the first 24 hours and does not recur later Gusev E.I., Burd G.S., Nikiforov A.S., 1999; Avakyan G.N., Generalov V.O., Oleinikova O.M. et al. 2004, Krylov V.V., Puras Yu.V. et al., 2010; Posner E., 2008).

Post-traumatic epilepsy is a chronic brain disease that occurs as a result of a traumatic brain injury within 3 to 18 months after a traumatic brain injury and is characterized by repeated unprovoked epileptic seizures. (Geht A.B., Kurkina I.V., Lokshina O.B. et al. 1999, Likhterman L.B., 2009, Thom M., 2003). The prevalence of epilepsy is about 50 cases per 100,000 population, including The most important causes of epilepsy include TBI (Karlov V.A., 1999; Konovalov

A.N., 1998; Yarmukhametova M.R., Bogdanov E.I., 2010; MacDonald BK , CockerelO . C. , SanderJ . W. etal . , 2000; JefferE ., 2003; ThomM ., 2003).

Post-traumatic epilepsy (PTE) accounts for 5% of all cases of epilepsy and 2-28% of cases of epilepsy that occurred after TBI, of varying severity (Kravchuk A.D., Okhlopkov V.A., 1998; Starodubtsev A.A., 2010; Friedman H., 2001; Mazarati A., 2006).

1.2. Traumatic seizures and post-traumatic epilepsy

During post-traumatic paroxysms, one should distinguish between post-traumatic epilepsy and post-traumatic epileptic seizures (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Krylov V.V., Talypov A.E., Puras Yu.V. and et al., 2010; Posner E., 2008). According to V.V. Krylova et al. (2010), any epileptic seizure that developed as a result of a traumatic brain injury should be attributed to a post-traumatic epileptic seizure.

Attacks that occur in the first 24 hours after a head injury are usually referred to as immediate, during the first week - early e , which occurred a week later - late (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Pagni C.A. , 2005). According to S. A. Pagni (1993), M. Swash (1998), the frequency of early post-traumatic seizures is 3-5%, late - 8-9%. Late post-traumatic seizures are characterized by a high recurrence of paroxysms (Haltiner AM, 1997). Approximately 1/3 to 2/3 of people with post-traumatic epilepsy experience a first seizure within the first 12 months after a brain injury and more than 75% at the end of the second year after TBI. Since many of the risk factors for PTE and early post-traumatic seizures are the same, it is difficult to determine whether the occurrence of early post-traumatic seizures is a risk factor for the development of PTE. However, even regardless of other common risk factors, in the presence of early post-traumatic seizures, an increase in the risk of developing PTE by more than 25% was found (Starodubtsev A.A., 2010; Beghi E., 2004; D, AmbrosioE ., 2004; ManiJ . , 2006 ; SanderJ.W . , 2007) .

Risk factors by sex and age are as follows: PTE occurs in 10% of children and 16-20% of adults after severe TBI. Persons over 65 years of age are at greater risk of developing epilepsy after TBI. Early post-traumatic seizures are more common in children under 5 years of age. The incidence of PTE is higher among men (Diaz-Arrastia, 2000; Herman ST, 2002; TuckerG . J. , 2005; PitkanenA ., 2006).

The incidence of PTE also depends on the severity of TBI: the more severe the TBI, the higher the risk of developing PTE (D, Ambrosio M., 2004). The risk of developing PTE in people with mild TBI is 1.5 times higher than in healthy people. According to some estimates (Iudice A., 2000), at least half of people who have received severe TBI have PTE, according to other data (Oliveros-Juste A., 2002), the risk of developing PTE after severe TBI is only 15-20%. The development of PTE 30 years after TBI is possible in 2.1% after mild TBI, in 4.2% after moderate TBI, and in 16.7% after severe TBI (Annegers J. F., 1998). Repeated surgical interventions, cerebral edema (Agrawal A., 2006), intracranial hematomas (Frey LC, 2003), intracranial hemorrhage (Kollevold T., 1978), cerebral contusion (Helmut W., 2002), depression bone fragments (Wiederhold, 1989). Subdural hematomas confer a higher risk of PTE than epidural hematomas due to greater brain tissue damage (Agrawal A., 2006). In addition, the risk of developing PTE is determined by the localization of traumatic brain injury : in case of brain contusion in one of the frontal lobes, the risk of developing PTE is 20%, in

one of the parietal lobes - 19%, in the temporal lobe - with bruises in both hemispheres, the risk of developing PTE is 26% for the frontal lobes, 66% - for the parietal and 31% - for the temporal 15 (GrafmanJ ., JonasB . , SalazarA . , 1992 ; GargaN ., LowensteinD . H. , 2006 ; PitkanenA ., 2007).

Risk factors for the development of PTE are also chronic alcoholism, prolonged post-traumatic amnesia, loss of consciousness, local neurological deficit, brain contusion, epileptic seizure within the first 24 hours after TBI, coma lasting more than 24 hours, status epilepticus (Heikinnen E., 1990;

Willmore L. J., 1990; Pechadre J. C., 1991; David W. , 2003; FirlikK ., 2004; PosnerE ., 2008).

Epileptic seizures are more common in children aged 3 to 14 years. Seizures are not uncommon in TBI in young and middle-aged patients, and are extremely rare in the elderly. The average age of patients with epileptic seizures due to TBI is 30 years. (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; HahnY . S ., FuchsS ., FlanneryA . M . et al., 1988; Annegers JF, Hauser A., Coan SP et al. ., 1998).

Symptomatic post-traumatic epilepsy develops in 11-20% of those who have undergone TBI; its frequency and severity depend on the severity of the primary TBI, localization of the post-traumatic focus, the state of the premorbid background (presence of repeated TBI and genetic predisposition) [Gusev E.I., Konovalov A.N., Skvortsova V.I., 2010; Krylov V.V., 2010; Lo Y., Yiu CH, HuH . H. , 1994; D , AmbrosioE ., 2004]. The critical time for the formation of post-traumatic epilepsy is considered the first 18 months after the injury. Epileptic seizures with intracranial hematomas occur about 10 times more often than with brain contusions. In adult patients, early epilepsy develops, usually in the presence of a depressed skull fracture or intracranial hematomas. Late epilepsy is 8-10 times more likely to occur after a penetrating brain injury than after a closed TBI. The timing of the onset of seizures after TBI is different: from 6 months to one year; during these periods, seizures occur in 72% of patients with post-traumatic epileptic syndrome. The rest of the seizures appeared in the period from one to 9 years. (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Krylov V.V., 2010). M.M. Odinak, V.A. Khilko, A.Yu. Emelyanov (2000) believe that post-traumatic epilepsy is detected in 13% of patients with moderate brain contusion, in 41% of patients with severe TBI and in 45% with open TBI. Damage to the meninges during brain injury increases the likelihood of developing epilepsy up to 36-53% (Gusev E.I., 2004).

1.3. PATHOGENESIS OF POSTTRAUMATIC EPILEPSY .

The main substrate of post-traumatic brain injuries is focal damage to neurons, which is morphologically manifested by hypoxic and sclerotic changes in the brain tissue, the formation of collagen, glial and agyrophic brain scars and cerebrospinal fluid cysts. Post-traumatic scars and liquor cysts, not being nervous tissue, cannot be conductors and generators of bioelectric activity. The formation of an epileptic focus occurs in adjacent areas of the brain that have undergone post-traumatic morphofunctional restructuring (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Shtulman D.R., Levin O.S., 2005). The basis of the occurrence of epileptic seizures is a trigger mechanism, the carrier of which is a population of neurons with special pathophysiological properties. These are the so-called epileptic neurons. Along with this fundamental factor, the specific mechanisms of epilepsy can differ significantly in different types of epilepsy. In generalized epilepsy, the main role in the work of the trigger mechanism is assigned to the nonspecific nuclei of the thalamus opticus. With symptomatic

epilepsy, the main role belongs to neurons located in the area of epileptogenic lesions and usually located on its periphery. Epileptic neurons in their totality form an epileptic focus, in which certain morphological changes are also noted: the absence of dendritic spines, depletion of axosomatic synapses, sprouting, etc., as well as changes in glia. These changes are also considered as a manifestation of partial neuronal deafferentation, which can explain both increased spontaneous activity and hypersensitivity of synaptic receptors.

At the same time, there are signs of massive activation of axodendritic synapses. The cardinal property of epileptic neurons is the paroxysmal depolarization shift of the membrane potential and their associated tendency to depolarization, i.e., excitation. To explain this phenomenon, three concepts are involved: 1) epileptic neuron, according to which damage in the membrane of the neuron leads to its hypersensitivity; 2) epileptic environment - dysregulation of the concentration of extracellular ions, transmitters, or both, leading to their

imbalance and increased neuronal excitability; 3) neuronal population, implying massive anatomical and/or functional alteration of neurons. At present, it is obvious that all the above violations are taking place. At the same time, not only neurons, but also glial cells are involved in epileptogenesis, although they do not initiate seizures (Yakhno N.N., 2007; Likhterman L.B., 2009; Mishnyakova L.P., 2008; Plum F., Posner JB, 2007).

At the level of a neuron, the process of excitation is associated with an excitatory presynaptic potential, while inhibition is associated with an inhibitory one. Neurotransmitters are involved in their generation: glutamatergic - excitatory and GABAergic inhibitory systems. There are three types of glutamate receptors, the most important of which is NMDA (N-methyl-D-aspartate) - subtype; excessive activation of NMDA receptors leads to an imbalance of excitation / inhibition with a predominance of excitation. NMDA - a activation is attributed a special role in epileptogenesis, since the NMDA system is inactivated by resting potentials and activated by the potentials of an already excited neuron (Karlov V.A., 1990). paralysis, a deficiency of NAMK-ergic inhibition of interneurons is found. On the other hand, excessive hyperpolarization, induced by epileptic spike activity and represented by a slow wave following the spike, is of primary importance in the mechanism of absence forms of epilepsy, forming the rhythm of spike-wave discharges. Thus, in these forms of epilepsy, there is no deficiency of the GABAergic system; moreover, in some experimental models of epilepsy, an increase in GABAergic terminals has been shown. Important in the development of polarization and hyperpolarization is the activation of the ion pump, whose activity is associated with the use of energy. Therefore, metabolic insufficiency, caused, for example, by hypoxia or ischemia of neurons, may be accompanied by a predominance of depolarization, i.e. excitation, and thus underlie epileptogenesis. An imbalance in other biochemical systems, in particular catecholamines and neuropeptides, can also have a certain significance in epileptogenesis (Yakhno N.N., 2007). All these changes occur primarily in the epileptic focus, which is characterized by special

pathophysiological properties - synchrony and in-phase discharges of epileptic neurons , as a result of which the discharge discharged from it is hypersynchronous. However, the presence of an epileptic focus is still not enough for the development of epilepsy. Its activation and the spread of epileptic activity beyond its limits, i.e., the formation of an epileptic system, are necessary. This is prevented by the defense mechanisms of the brain - the anti-epileptic system. It is induced by the epileptic focus itself according to the negative feedback mechanism: recurrent inhibition of neurons around the focus, activation of certain structures that have an inhibitory effect on the epileptic focus. The antiepileptic system includes the reticular nuclei of the pons, the cerebellum, the caudate nucleus, the orbitofrontal cortex (Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010).

The balance of epileptogenic / antiepileptogenic influences varies depending on a number of factors: the basic functional state of the brain (wakefulness, slow-wave and fast-wave sleep), hormonal changes (menstrual cycle, puberty , menopause), the effects of mental and other factors (especially sleep deprivation , alcoholism). This can lead to activation of the epileptic focus and a breakthrough of epileptic activity outside the focus, i.e., to the development of a seizure. The activation of the focus consists in the involvement in the epileptic excitation of neurons that are in the focus, but do not have their own epileptic activity. As a result, the number of synchronously and in-phase discharge neurons can reach a "critical mass" with the spread of epileptic activity to other cerebral structures, including generalizing formations of the subcortex (secondary recurrent generalization), and partial and secondary generalized seizures occur. At the same time, other forms of spread of epileptic activity are also possible: by involving interneurons, as well as through the commissures of the brain, mainly transcallosally (Bein B.N., 2010). The role of the ephaptic, i.e., extrasynaptic mechanism , especially in the hippocampus, is also not excluded (Karlov V.A., 2000, 2007).

Currently, the concept of a two-stage epileptogenesis is being expressed. The first stage is from brain damage to the development of the first epileptic seizure. This is the stage of maturation of the epileptic focus. The second stage begins after the clinical debut of the disease - an epileptic seizure. At this stage, the spread of epileptic activity beyond the focus increases synaptic conductance in the pathways of its propagation, lowers the excitation threshold of neurons, facilitating the development of subsequent seizures, in other words, a stable epileptic system of partial epilepsy is formed. Another type of epileptic system occurs in generalized epilepsy. Here the question of the epileptic focus remains open. Modern examination methods show that, as a rule, neural ectopias are the basis as a result of microdysontogenesis. Due to the violation of neuronal synaptic connections, deafferentation hypersensitivity of neurons occurs. A known role may be played by the constitutional insufficiency of some biological systems, in particular, catecholamines. The pacemaker of epileptic activity is the nonspecific nuclei of the thalamus with thalamocortical and reverse corticothalamic circles of circulation of epileptic excitation. There is also a decrease in the tone of the cerebral cortex and an increase in the excitability of cortical neurons (Yakhno N.N., 2007).

Clinical and electroencephalographic comparison shows that generalized epilepsy is characterized by two patterns: an absence with generalized symmetrical activity with a frequency of 3 per second and a primary generalized tonic-clonic seizure (GTCS), debuting at a rhythm of about 10 per second.

Epileptic systems as a whole are characterized by the complexity of organization and hierarchical relationships within them, dynamism and gradual complication in the course of the disease. Under the influence of epileptic "bombardment", secondary and even tertiary foci are formed from the epileptic focus in the cerebral cortex . At first, they are dependent and may disappear with the elimination of the primary focus, but later they become independent. In general, the process of epileptization can gradually involve the entire brain. The epileptic focus not only disorganizes the activity of the brain, but also

reorganizes it in a special epileptic way, actually controlling the functional state of the brain.

Functional depletion of interneurons plays an important role. Constant epileptic activity requires increased energy expenditure. Epileptic foci in the interictal stage are characterized by hypometabolism, in particular, due to insufficient blood flow at the capillary level. Within a certain time, metabolic insufficiency is compensated to one degree or another. In this process, a certain role is played by glia, which intensively synthesizes reserves of protein and RNA for the neuron in the cytoplasm (these processes in glia are anaerobic). However, the trophic supply of epileptic activity simultaneously contributes to the progression of brain epileptization. Ultimately, depletion of the functional capabilities of glia occurs, gliosis develops, thickening of the brain tissue and other disorders occur (Skoromets A.A., Skoromets A.P., Skoromets T.A., 2005; Shtulman D.R., Levin O.S. ., 2005; Mazarati A., 2006).

The starting point for the development of post-traumatic epilepsy is the foci of primary damage that occurs mainly in the frontotemporal regions of the brain. Morphological changes in the zone of the epileptic focus vary from ultrastructural to rough adhesive-scar zones. However, the presence of such a traumatic brain injury cannot be considered sufficient for the development of PTE (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; GrafmanJ ., JonasB ., SalazarA ., 1992). For the development of epilepsy, it is necessary to have a persistent focus of epileptic activity due to organic damage to the brain. At the same time, the epileptization of neurons, their special condition, which determines the " road readiness" of the brain in the foci of its organic lesion and the degree of epileptic influence of these foci on brain structures, also depends on the premorbid characteristics of the organism, in particular, on epileptic predisposition, genetic or acquired nature. , which determines the greater likelihood of an epileptic seizure in a patient with brain damage (Yarmukhametova M.R., 2010).

Currently, there are several hypotheses for the development of post-traumatic epilepsy. In the period between traumatic brain injury and the appearance of recurrent epileptic seizures in particularly vulnerable areas of the cortex, hippocampus, damaged brain cells can form new synapses and axons, undergo apoptosis or necrosis (Konovalov A.N., Likhterman L.B., Potapov A. A., 1998; Mani J., 2006). In addition, the particularly vulnerable position of the cortical regions, the hippocampus, can lead to PTE. Blood accumulated in the brain after an injury can cause damage to brain tissue and thus cause epilepsy. Products that are formed as a result of the breakdown of blood hemoglobin can be toxic to brain tissue. PTE occurs as a result of oxygen damage by free radicals, formations that are catalyzed from blood iron. Iron is catalyzed by the formation of hydroxyl radicals, which damage brain cells by lipid peroxidation in membranes. TBI can lead to excessive release of glutamate and other neurotransmitters. This excessive release of glutamate can cause excitotoxicity - damage to brain cells through greater activation of receptors that bind and respond to neurotransmitter stimuli. Overactivation of glutamine receptors leads to damage to neurons, the formation of free radicals.

Excitotoxicity is a possible factor in the development of PTE and may lead to the formation of a chronic epileptogenic focus. Seizures that occur shortly after a TBI can reorganize neural networks and cause seizures that later occur continuously and spontaneously. New neural connections form in the brain and cause an increase in excitability, and this reorganization of the neural network can make them more excitable. Neurons that are over-excited due to injury can create an epileptic focus in the brain, resulting in seizures. An increase in neuronal excitability may be accompanied by a loss of inhibitory neurons, leading to the development of PTE. Early attacks are likely to differ from late ones in pathogenesis. Late post-traumatic seizures are unprovoked, while early ones are the result of direct exposure.

on the brain, acute reaction of cortical brain damage. The degree of hydrocephalus and hypoperfusion in the parietal lobe is a significant risk for the

development of late post-traumatic seizures . Local, tissue destruction is an important factor in predicting the development of late post-traumatic seizures (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998 ; Krylov V. V., Talypov A. E., Puras Yu. V. et al., 2010; Abel MS, 1992; Letizia M., 2003; Thom M., 2003; Agrawal A., 2006; GuptaY . K. , 2006 ; PosnerE ., 2008). V.V. Krylov, A.E. Talypov, Yu.V. Puras, I.S. Trifanov (2010) indicate that the volume of the bruise does not have a significant effect on the development of post-traumatic epilepsy, the localization of the focus of the bruise and surgical treatment play an important role. Early and late epileptic seizures developed only in patients with small-focal brain contusions, around which perifocal edema, which is an epileptogenic zone, persisted for a long time. The pathogenesis of the development of early and late epileptic seizures in TBI is different. The occurrence of early seizures is due to cytotoxic and metabolic changes in the focus of brain damage, as well as the compression effect of the traumatic focus on brain structures. Late epileptic seizures are associated with the gradual formation of an epileptic focus (Gusev E.I., Konovalov A.N., Skvortsova V.I. et al., 2010; Starodubtsev A.A., 2010; Hauser W . A ., Annegers J . F ., Kurland L. T. , 1993). A higher probability of developing epilepsy in the late period of injury is determined by the following factors: prolonged coma of the patient (more than 24 hours); damage to the motor area of the cerebral hemisphere, mediobasal parts of the frontal and temporal lobes of the brain; the presence of depressed skull fractures; perinatal pathology and history of alcohol abuse (Makarov A.Yu., 2001; Potapov A.A., Likhterman L.B., Vos P.E. et al., 2002; Alekseenko Yu.V., 2006; Annegers JF, Hauser WA, Coan SP, 1998; FergusonP . L. , SmithG . M. , WannamakerB . b . et al ., 2009).

1.4. CLASSIFICATION OF EPILEPTIC SEIZURES

Epileptic seizures are the leading symptoms of irritation in the clinical picture of TBI. For their terminological unification, it is necessary to use the

international classification of epileptic seizures (Zenkov L.R., 2010; Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010; Kissin M.Ya.; Pellock JM, 1989).

In 1981, the Commission on Terminology and Classification of the International League Against Epilepticism adopted an international classification of epileptic seizures, according to which partial (focal, local) and generalized seizures are distinguished.

I. Partial seizures are divided into simple, complex (occurring with impaired consciousness) and secondary generalized.

* Simple (not accompanied by loss of consciousness) partial seizures:

- with motor signs;

- with somatosensory (sensation of numbness, passage of "current" in the contralateral focus of the extremities or half of the face) or specific sensory symptoms (simple hallucinations, such as sounds, flashes of light or lightning);

- with autonomic symptoms or signs (peculiar epigastric sensations, pallor, sweating, skin redness, piloeresis, mydriasis);

- with mental symptoms.

* Complex partial seizures are accompanied by a change in consciousness : the beginning can be with a simple partial seizure followed by impaired consciousness or impaired consciousness in an attack (temporal pseudo-absences and automatisms).

II. In primary generalized seizures, both hemispheres of the brain are initially involved in the pathological process. There are the following types of generalized seizures:

* absences and atypical absences;

* myoclonic;

* clonic;

*tonic;

* tonic-clonic;

*atonic.

III. Unclassified epileptic seizures (seizures that

cannot be included in any of the above groups due to the lack of necessary information, as well as some neonatal seizures, for example , rhythmic eye movements, chewing, spitting movements).

IV. Repeated epileptic seizures (random, cyclic, provoked).

V. Prolonged seizures (status epilepticus). (Yakhno N.N., 2007, Gusev E.I., Konovalov A.N., Skvortsova V.I. et al., 2010)

The International Classification of Epilepsy, adopted in 1989 by the International Anti-Epileptic League, is based on 2 principles. The first is to determine whether the epilepsy is focal or generalized. According to the second principle, idiopathic, symptomatic or cryptogenic epilepsy is distinguished.

1. Localized (focal, local, partial) epilepsy:

*idiopathic;

*symptomatic (frontal, temporal, parietal, occipital lobe epilepsy);

* cryptogenic.

2. Generalized epilepsy:

* idiopathic (including childhood and juvenile absence epilepsy);

*symptomatic;

*cryptogenic.

3. Nondeterministic epilepsy (Gusev E.I., Burd G.S., Nikiforov A.S., 1999; Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010).

In 2001, the International Commission on Classification and Terminology released a new classification of epileptic seizures. It has not yet received final approval, but is currently recommended for use in clinical practice. The classification is based on classical concepts of focal and generalized forms of epilepsy. Diagnose focal seizures and focal epileptic syndromes if the nature of paroxysms, EEG data and neuroimaging methods confirm the local nature of epileptic seizures. With focal paroxysms, the concept of a cortical "epileptogenic focus" has been created, which plays the role of a "pacemaker". A hypersynchronous discharge from an epileptogenic focus involves a large

number of gray matter neurons and spreads to certain areas of the brain. With generalized forms of epilepsy, seizures should be generalized from the very beginning, which is also confirmed by EEG data. The pathogenesis of generalized forms of epilepsy is still not clear enough .

A cortico-thalamocortical hypothesis of the origin of primary generalization has been put forward . According to the classification, paroxysmal episodes are diagnosed as epileptic if their cortical origin is proven due to the occurrence of hypersynchronous neuronal discharges. Diagnosis of epilepsy is established only if epileptic seizures recur and the disease satisfies the definition given above.

The latest classification introduced significant innovations, primarily terminological. The term "partial seizures and partial epilepsies" has been replaced by "focal seizures and focal epilepsies". The definition of "cryptogenic forms" was replaced by "probably symptomatic forms". In the definition of syndromes, it is recommended to replace the word “convulsions” with “attacks”. The concept of "seizures" is much broader than "seizures", and not all seizures are manifested by convulsions. The division of focal seizures into simple and complex ones, depending on the level of impaired consciousness, has been abolished. This is due to the fact that in most cases the doctor fails to test the patient's consciousness in detail during an attack, and therefore the assessment of the level of consciousness is always indicative (Karlov V.A., 2007; Mukhin K.Yu., Petrukhin A.S., Mironov M. .B., 2008; Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010).

The same commission proposed the following classification of epileptic seizures.

I. Self-limiting seizures:

* tonic-clonic (including options for starting with a clonic or myoclonic phase);

* clonic (with or without a slight tonic component);

*typical absences;

* atypical absences;

* myoclonic absences;

*tonic;

* epileptic spasms;

*epileptic myoclonus;

* eyelid myoclonus (with or without absences);

*myoclonic-astatic (myotonic);

*negative myoclonus;

*atonic;

* reflex generalized.

II. focal:

* focal sensory (with simple symptoms associated with irritation of the occipital or parietal lobe, or with complex symptoms associated with irritation of the temporo-parietal-occipital cortex);

* focal motor: clonic, asymmetric tonic (associated with irritation of the additional motor zone), with typical automatisms , with hyperkinetic automatisms, with focal negative myoclonus, inhibitory;

* gelastic;

* hemiclonic;

*secondary-generalized;

*reflex focal.

III. Ongoing seizures:

* generalized status epilepticus:

- status of generalized tonic-clonic seizures,

- status of clonic seizures,

- status of tonic seizures,

- status of myoclonic seizures;

* focal status epilepticus:

- Kozhevnikov epilepsy,

- extended aura

- status of limbic seizures (psychomotor status),

- hemiconvulsive status with hemiparesis.

IV. Provoking factors in reflex seizures:

* visual stimuli: flickering light (it is desirable to indicate the color);

*thinking process;

*music;

*food;

* performance of movements;

* somatosensory stimuli;

* proprioceptive stimuli;

* reading;

* hot water;

* sharp sound (startl-attacks). (Karlov V.A., 2002; Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010; Chorvon SD, 1990).

When assessing the timing of the occurrence of traumatic epileptic seizures, they are most often divided into :

- early, developing in terms from 0 to 7 days from the moment of the disease;

- late , arising after 7 days or more (Gusev E.I., Konovalov A.N., Skvortsova V.I., Gekht A.B., 2010; Krylov V.V., Talypov A.E., Puras Yu V., 2010, Barolin G. S. , SherzerE , 1962).

In addition, some authors (Krylov V.V., Talypov A.E., Puras Yu.V., Trifonov I.S., 2010; Samuels, 1999; Temkin NR, 2003) indicate the need to isolate more immediate epileptic seizures developing in the first 24 hours after injury, emphasizing their important prognostic role in the development of post-traumatic epilepsy . Thus, according to N. Temkin, the risk of developing PTE in victims with TBI and a history of immediate epileptic seizures is 28%. According to CA Pagni (1993), the frequency of early post-traumatic seizures is 3-5%, late - 8-9%. Late post-traumatic seizures are characterized by a high frequency of paroxysms (Haltiner AM, 1997). R. D, Ambrosio (2004) points out that many of the risk factors for post -post-traumatic epilepsy and early post-traumatic seizures are the same, it is not known whether the occurrence of early

post-traumatic seizures is a risk factor for the development of post-post-traumatic epilepsy.

1.5. CLINICAL PICTURE OF POST-POSTTRAUMATIC EPILEPSY

Traumatic epilepsy is characterized by a variety of clinical forms, largely associated with the features of brain damage (concussion, contusion, compression). According to A.N. Konovalova, L.B. Likhterman , A.A. Potapova (1998) in patients with bruises, the focal type of epileptic seizures predominates; in patients with concussion and brain compression - generalized (Cuccurullo S., 2004) and secondary generalized (Posner E., 2008) type of seizures.

Every third patient with PTE has partial seizures, which can be simple or complex (Parent JM, 2005). With simple partial seizures, the level of consciousness does not change, while with complex ones, a violation of consciousness is noted (Ayd F. J., 2000). In generalized seizures, they start as partial and then spread and become generalized. Most early post-traumatic seizures are partial , while most late post-traumatic seizures are primary or secondary generalized. According to PM Vespa (1999), 52% of patients with post-traumatic epilepsy also have non-convulsive seizures.

In the neurological status of a patient with PTE, disorders caused by a previous TBI are revealed. In the presence of a motor or sensory deficit in the neurological status, a partial onset of an attack is noted on the side of the affected limbs. It is not uncommon for several years to elapse between trauma and the onset of post-traumatic epilepsy. The presence of a focal brain lesion on neuroimaging correlated with changes in the EEG and the semiology and kinematics of seizures suggests a traumatic etiology of epilepsy. The delayed onset of seizures is associated with the initially high capabilities of the antiepileptic system of the brain and a decrease in its
activity under the influence of vascular, toxic and age factors (Gusev E.I., 2004).

Differences of post-traumatic epilepsy from other symptomatic forms are: less pronounced progradient, dependence of the course of the disease on the severity of TBI, a combination of epileptic seizures with persistent focal neurological symptoms, often deepening after a seizure for a period of several minutes to several hours, the prevalence of focal manifestations in the structure of an attack, less pronounced than in other types of symptomatic epilepsy, the progression of intellectual-mnestic disorders. (Gusev E.I., 2004, Posner E., 2008). The localization-conditioned nature of PTE determines the predominance of various types of partial seizures in the epilepsy clinic. The kinematics of the seizure correlates with the localization of the epileptogenic focus, which leads to a diverse semiology of seizures in post-post-traumatic epilepsy.

Simple partial seizures are epileptic paroxysms that occur without impairment of consciousness and are characterized by various symptoms (motor, sensory, mental, vegetative, combined) in the presence of an epileptogenic focus in certain areas of the cerebral cortex.

Motor partial seizures - Jacksonian, adversive, postural, phonatory . D Jacksonian motor seizures are characterized by local convulsions on the side opposite to the pathological focus when the epileptic focus is localized in the precentral gyrus of the cerebral hemisphere. deviation of the head and eyes in the direction opposite to the epileptic focus. Localization

epileptic focus in the posterior sections of the second frontal gyrus. Phonator seizures are motor partial seizures characterized by involuntary vocalization or speech cessation.

Sensory seizures: somatosensory, visual, auditory, olfactory, gustatory, bouts of vestibular vertigo.

Somatosensory , also called sensory Jacksonian

paroxysms, manifested by paroxysms of paresthesias in the form of tingling, numbness, crawling, occur on the opposite side of the body to the epileptogenic focus (leg, torso, arm, face) and subsequently can spread throughout the entire

half of the body like a Jacksonian march. The localization of the epileptic focus is the postcentral gyrus.

Visual partial seizures associated with a focus of epileptic activity in the occipital cortex are manifested by flashes of light, lightning, color balls, spots, cattle, hemianopsia or cortical blindness lasting a second, less often minutes, and with a focus in the occipitotemporal cortex - simple hallucinations.

Simple partial seizures with gustatory, olfactory disturbances are characterized by a change in taste sensations . With auditory partial paroxysms, they are characterized by stereotype and short duration of symptoms and vary from simple to complex auditory hallucinations.

Complex partial seizures differ from simple ones in more pronounced clinical manifestations, and most importantly, in a violation of consciousness: the patient is aware of the attack, but cannot respond to the environment (does not follow commands, does not answer questions), or, conversely, can do this, but automatically, not realizing what is happening (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Yakhno N.N., 2007; Likhterman L.B., 2009) . partial seizure. First of all , this is a feeling of the unreality of what is happening, the alienation of the outside world (derealization) or unreal feelings inside oneself (depersonalization). Seizures with cognitive symptoms include seizures with ideational, dysmnestic, and other disorders. The most common ideational phenomenon is forced thinking - attacks of obsessive thoughts: subjective (for example, thoughts about death) or objective (fixation on a previously heard phrase).

A dysmnesic seizure is characterized by paroxysmal memory disorders, in particular, violent memories in the smallest detail of the events of a previous life, states already seen (the new environment seems already familiar), never seen (the familiar environment seems unfamiliar), already experienced, never experienced, etc. These conditions are usually combined with affective changes of a negative nature (sadness, anxiety) and most often occur with temporal lobe epilepsy in the case of right-sided foci in right-handers. On the contrary, the

state of the already heard, never heard is characteristic of the left-sided lesion of the temporal lobe, since they usually belong to the auditory-speech sphere (Yakhno N.N., 2007; Likhterman L.B., 2009).

Another striking manifestation of a complex partial seizure is epileptic automatism - involuntary motor activity, more or less coordinated and adapted, manifested during or after a seizure, usually accompanied by amnesia (psychomotor seizure). Epileptic automatism can be a sign of both a partial seizure with a temporal or frontal focus, and a generalized epileptic seizure - an absence of automatisms. There are automatisms of eating (chewing, licking, swallowing), mimic, reflecting the emotional state of the patient (for example, fear), gesticulation, verbal and outpatient. With the latter , the patient moves on foot or by transport for one or another distance. True epileptic seizures of automatism are short-term (minutes). Longer ambulatory automatisms are the result of post-seizure confusion or epileptic status of psychomotor seizures (Karlov V.A., 2000).

Any partial seizure, simple or complex, can progress to a generalized seizure (secondary generalized

seizure). In cases where the patient retains memories of the sensations of the beginning of the seizure before losing consciousness, they speak of the aura of the seizure. The aura is part of the seizure, not a symptom that precedes it. The aura is called a symptom-signal, since with a generalized convulsive seizure, the aura indicates partial epilepsy with secondary generalization and allows you to establish the location of the focus. Accordingly, the aura can be motor, sensory, sensitive (visual, auditory, olfactory, gustatory), mental, vegetative. Harbingers of an epileptic seizure should be distinguished from the aura - certain sensations characteristic of a given patient that occur over a different period of time (hours, rarely a day) before the seizure. Most often, these are sleep disorders, anxiety or depression, indicating a growing imbalance of the epileptic and antiepileptic systems and an impending seizure (Yakhno N.N., 2007).

Secondary-generalized partial seizures begin as simple or complex-partial seizures, later transforming into a general convulsive seizure . Generalized seizures are characterized by: 1) clinically - loss of consciousness, massive vegetative manifestations, accompanied or not accompanied by convulsive manifestations involving both sides of the body at the same time; 2) electroencephalographically - generalized synchronous symmetrical discharges of an epileptic seizure. Accordingly, there are two types of generalized epileptic seizures - convulsive and non-convulsive.

Generalized tonic-clonic seizures are the most common form of convulsive epileptic seizures. It begins abruptly with a brief initial phase (seconds) during which loss of consciousness occurs and slight bilateral myoclonic twitches develop, usually unnoticed. Already in this phase, pupil dilation is observed, followed by a phase of tonic convulsions (tens of seconds): convulsions cover the entire skeletal muscles - the eyes are usually wide open, the eyeballs diverge and roll up. Then tonic convulsions are replaced by clonic ones, while convulsions alternate every few seconds with muscle relaxation periods. The latter gradually lengthens, after 30-40 s the clonic phase ends and the postictal period begins, which can be represented by a coma, turning into sleep, or short-term stunning, or, finally, psychomotor agitation. As a result of apnea in the tonic stage, cyanosis occurs, the veins in the neck swell, the carotid arteries pulsate intensely (Gusev E.I., Konovalov A.N., Skvortsova V.I., 2010). also hypersalivation, which, in combination with a bite of the tongue and convulsions, is accompanied by the release of bloody, foamy sputum from the mouth. Often during a seizure, patients are seriously injured. The electrographic correlate of GTCS are: in the initial phase - a monomorphic low-amplitude rhythm, with a frequency of about 10 Hz, in the tonic phase - a rapid increase in the amplitude of the rhythm (the phenomenon of recruitment, i.e., the involvement of an increasing number of neurons in the discharge); in the clonic stage, a slowing rhythm of spike- wave. An attack can end in two ways: a flattening of the curve or slow-wave oscillations, which reflects the presence of

two mechanisms for stopping the seizure - passive (metabolic exhaustion) and active - inhibition through inhibition. In some cases, seizures can only have a tonic or only clonic character . In most cases, the EEG corresponds to the tonic and clonic stages of GTCS.

Myoclonic epileptic seizures, myoclonus caused by an epileptic discharge, are also most common in children and adolescents. They can be both generalized and limited, in the area of the face and upper limb, or involve only one or a few limbs. Usually , epileptic myoclonus is of cortical origin, and therefore, consciousness can remain preserved with them. On the EEG, myoclonus corresponds to spikes or polyspike waves, sometimes followed by a slow wave. There is a large group of different forms of myoclonic epilepsy . Massive myoclonus or muscle atony that occurs in the pauses between them can cause falls and injuries to the child (Grindel O.M., 1998; Likhterman L.B., 2009)

Another type of generalized epileptic seizures are non-convulsive seizures - absences. This type of seizure usually occurs in childhood. During a seizure, the patient is unconscious, the current activity is interrupted, and he is likened to a statue with an empty look. This picture corresponds to a simple absence. The patient is not in contact, they do not feel a seizure, especially since it lasts a maximum of 10-20 s. In the EEG, the picture of a typical absence corresponds - a generalized, synchronous, symmetrical spike-wave activity - 3 Hz, slowing down during a seizure to 2.5-2 Hz. False absence is a simple absence plus various motor phenomena: for example, myoclonic twitching of the eyelids , muscles of the face, muscles of the girdle of the upper extremities (myoclonic absence); elementary automatisms - muttering, fingering, etc. (absence of automatisms) or falling, due to the switching off of the postural tone (atonic absence). The electrographic picture may correspond to a typical absence or atypical - generalized, synchronous, sometimes asymmetric in amplitude, spike-wave activity with a frequency of less than 3 Hz (2.5-1.5 Hz), sometimes 4 Hz

or more (Karlov V.A., 1999; Skoromets A.A., Skoromets A.P., Skoromets T.A., 2005).

Most early post-traumatic seizures are partial , while most late post-traumatic seizures are primary or secondary generalized (Gusev E.I., 2004; Posner E., 2008). In the presence of two or more epileptic foci due to brain damage, seizures may have a polymorphic character. The epileptic convulsive syndrome in TBI often has a clear cortical coloration. Partial seizures predominate. Their most common variant is faciobrachial: tonic-clonic contractions of the muscles of half of the face with spread to the arm of the same name, with a predominance of convulsions in the flexors. Adversive seizures are not uncommon, starting with a combined deviation of the eyes and head to the side opposite to the focus of damage or compression of the brain. Primarily generalized convulsive seizures are more often observed with mediobasal frontal and temporal lesions. (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998). In patients with severe TBI, tonic-clonic generalized epileptic seizures are most often observed, which lead to increased intracranial pressure, impaired cerebral perfusion, ischemia and increase in cerebral edema (Konovalov A.N. et al., 1997; Ferguson PL, 2009).

1.6. DIAGNOSTICS OF POSTTRAUMATIC EPILEPSY

To make a diagnosis of post-traumatic epilepsy, there must be two or more unprovoked seizures , in addition, there must be a mandatory connection between seizures and TBI, and it is necessary to exclude seizures caused by other factors, including fluid and electrolyte imbalances, hypoxia, ischemia, alcohol withdrawal. Thus, these factors should be excluded as the cause of seizures in people with TBI before diagnosing post-post-traumatic epilepsy (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Barry , E. 1997; FreyL . C ., 2003; Menkes J. , 2005; StatlerK . D. , 2006).

The most informative for the diagnosis of post-traumatic epilepsy is the clinical analysis of the structure of the seizure and the EEG, which reveals characteristic focal and cerebral irritative changes, as well as MRI. Subclinical epileptic disturbances on the EEG are focal peaks, “sharp” waves. These changes can regress, stabilize, or transform into an epileptic syndrome. The risk of developing post-traumatic epilepsy is high if peak-wave complexes, peaks or sharp waves replace focal delta activity or persist, increase for 3-6 months. and more after TBI (Grindel O.M., 1998; Zenkov L.R., 2009).

It should be noted that there is an opinion about the non-informativeness of visual EEG in the long-term period of TBI (Makarov A.Yu., Sadykov E.A., Kholin A.V., 2000).

Avakyan G.N., Generalov V.O., Oleynkova O.M. et. performing neuroimaging. They proposed the following score for the probability of PTE.

Symptoms	Points
Nature of injury 1 mild degree of craniocerebral injury 2 average degree of craniocerebral injury 3 severe degree of CTBI	 1 2 3
The duration of the blackout of consciousness during CTBI Less than 5 minutes From 5 - 10 minutes From 10 – 30 minutes	 0 1 2

30 minutes or more	3
Interval between injury and seizure onset 1 – 18 months 18 – 24 months Over 24 months	 3 2 1
Focal pathology on the EEG No one hearth more than one hearth	 0 2 3
post -traumatic changes Neuroimaging: not found on CT found on CT or MRI not found on MRI	 1 3 0
Electro-neuroimaging correlation within the lobe of the brain (grade 1) within the hemisphere (grade 2) no correlation (grade 3)	 3 2 1

The etiological relationship between trauma and epilepsy is very likely with a score of > 11, doubtful with a score of 7-10, unlikely with a score of < 7.

According to the recommendations of the Commission on Neuroimaging of the International League Against Epilepsy (ILAE), CT or MRI of the brain is indicated for all patients with the onset of epileptic seizures in the late period of TBI.

In post-traumatic epilepsy, neuroimaging is especially important, as it can act as an objective method for diagnosing morphological traumatic brain damage.

Carrying out CT scan of the brain in most patients with post-traumatic epilepsy reveals characteristic post-traumatic damage in the form of bone defects, cicatricial-atrophic lesions, post-traumatic cysts, post-traumatic hematomas, foci of reduced or increased density of deep and convexital localization (Kornienko V.N., Likhterman L.B. ., 1998; Likhterman L.P., 2008)

In post-traumatic epilepsy, an MRI study of the brain is preferable to CT, as it allows to detect microfocal changes either in the area of injury or in the contralateral hemisphere (according to the anti-shock mechanism) even in the absence of changes in the CT study (Kornienko V.N., Turkin A.M. ., Likhterman L.B., 1998; Vakulenko I.P., Gubenko O.V., Gulyameryants A.V., 2000).

When comparing the results of electroencephalographic and neuroradiological studies in patients with PTE, in most cases, the morphological post-traumatic focus correlates with electroencephalographic focal changes in bioelectrical activity. Correlation within the lobe of the hemisphere is found in most patients and is one of the objective criteria for post-traumatic etiology of epilepsy (Alikhanov A.A., 2001; Geibatova L.G., Karlov V.A., Gnezlinsky V.V., 2008).

When assessing the significance of the volume of morphological brain damage detected during neuroimaging, an inverse correlation is found - extensive post-traumatic changes are associated with later onset of seizures

compared to the onset of epilepsy in small and clearly defined areas of the brain parenchyma. In general, during screening examinations of persons who have suffered a traumatic brain injury, epilepsy is more often detected in persons with minor morphological changes, extensive brain damage is less likely to lead to the development of seizures. Convulsive seizures often join with convexital localization of the focus than in cases of its deep location (Alikhanov A.A., 2001; Alekseenko Yu.V., 2006; Yarmukhametova M.R., Bogdanov E.I., 2010).

Certain difficulties in diagnosing PTE in the acute period are due to the fact that paroxysmal conditions of various origins can be observed during this period (decompensation of concomitant somatic or neurological pathology, alcohol withdrawal, typical syncope, TIA, autonomic crises, psychogenic disorders). Most often they are non-epileptic in nature. (Konovalov A.N., Likhterman L.B., Potapov A.A., 1999; Barsukov A.V., Didenko M.V. et al., 2009).

The most likely is the development of PTE in the late period of TBI, which is characterized by such factors as prolonged coma, brain contusions in the motor and premotor zones, damage to the mediobasal parts of the temporal and frontal lobes, the development of predominantly subdural hematomas, penetrating brain injuries, the presence of early epileptic seizures, alcohol abuse (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; D, Ambrosio R., 2004; Mani J., 2006).

1.7. TREATMENT OF POSTTRAUMATIC EPILEPSY

Treatment of traumatic epilepsy is difficult and painstaking work, because antiepileptic therapy continues for a long period (3-5 years), and sometimes throughout life, with significant difficulties in the treatment process (Alekseenko Yu.V., 2006; Zenkov L.R. ., 2010; Yarmukhametova M.R., 2010; Garga N., 2006).

Currently, the "gold standard" in the treatment of epilepsy is monotherapy, and polytherapy has been used only when adequate monotherapy is impossible (Karlov V.A., 2010; Zenkov L.R.; Kissin M.Ya., 2011; Wolf P., 2003).

G.N. Avakyan, V.O. Generalov, O.M. Oleinikova, N.N. Maslova, O.L. Badalyan, S.G. Burd, A.N. Boyko (2004) c read that the treatment of PTE is an integral part of the complex rehabilitation of patients after TBI and consists of:

1. Treatment of traumatic brain disease, including correction of vascular, metabolic, autonomic, motor and other disorders caused by trauma;
2. anticonvulsant therapy;
3. Treatment of concomitant somatic pathology

The goal of antiepileptic pharmacotherapy, according to L.P. Zenkov (2010) is the complete cessation of seizures and the provision of pedagogical, professional and social adaptation of the patient.

Until recently, long-term use of antiepileptic drugs after TBI to prevent post-traumatic epilepsy was a standard common recommendation. Recent studies have shown that long-term prophylactic use of antiepileptic drugs in the acute period of TBI does not reduce the likelihood of further development of post-traumatic epilepsy, and from these positions, without taking into account additional factors and features of trauma, is not appropriate (Chang BS, 2003). At the same time, their administration in the acute period of injury (especially within 7 days) significantly reduces the risk of developing early epileptic seizures and can be recommended for 1-2 weeks for people with a high epileptic risk (with the development of intracranial hematomas, with penetrating and gunshot craniocerebral injuries, focal hemorrhagic bruises, depressed skull fractures, alcohol abuse and a history of epileptic seizures). It is also obvious that antiepileptic drugs are effective and necessary in the event of early epileptic seizures. And although the outcomes of TBI (mortality, disability) do not change significantly (Wiedemayer H., Triesch K., 2002), such a recommendation can be considered justified. Treatment of post-traumatic epilepsy is carried out taking

into account standard approaches to the treatment of epileptic disease (Avakyan G. N., Generalov V. O., Oleinikova O. M. et al., 2004; Zenkov L. R., 2010; Zelensky A. Yu., Kuprinenko N.V., 2010; Kissin M.Ya., 2011).

At the same time, the development of the first and only epileptic seizure, possibly of post-traumatic genesis, is a reason for appropriate examination and observation, but not yet for the appointment of epileptic therapy (Alekseenko Yu.V., 2006).

Drug treatment for developed post-traumatic epilepsy should be carried out continuously for several years, and sometimes throughout life. In the absence of epileptic signs for 2-5 years, normalization of the EEG, a gradual cessation of systematic antiepileptic therapy is acceptable (Konovalov A.N., Likhterman L.B., Potapov A.A., 1998; Likhterman L.B., 2009; Kissin M. .Ya., 2011).

The goal of anticonvulsant treatment in patients with epilepsy is to prevent the development of seizures in individuals who are prone to recurrence of epileptic seizures. The objective criteria for the effectiveness of the treatment regimen adopted by the patient is a decrease in the number of seizures over a certain period of time (week, month, year). Depending on the significance of this decrease, efficiency of 50% is distinguished - a decrease in the number of seizures by 50%, efficiency of 75% - a decrease in the number of seizures by 75%, efficiency of 100% - the complete absence of seizures. Another criterion for the effectiveness of treatment is the shortening of the unconscious state and post-seizure confusion, the transformation of generalized seizures into partial ones, which indicates a decrease in epileptic activity in the focus (Avakyan G.N., Generalov V.O., Oleynkova O.M. et al., 2004) .

The criteria for the risk of recurrence of epileptic seizures in persons who have undergone TBI is the detection of persistent regional EEG patterns correlated in terms of localization with morphological changes during neuroimaging.

Therefore, when these changes are detected, it is quite logical to prescribe anticonvulsant therapy after the first attack. The drug of choice for the treatment and prevention of post-traumatic epileptic seizures are valproates (depakin-chrono, etc.), carbamazepine, less often diphenin (the use of the latter is limited due to the risk of a more pronounced impairment of cognitive functions) [Posner E., 2008].

Most often, sodium valproate is recommended - Depakine Chrono in a daily dose of 600 mg, preferably after meals. It is possible to increase the dose by 200 mg every three days, but it is not recommended to exceed the daily dose of 2.5 g per day (Nikanorova M.Yu., Ermakov A.Yu.; Tucker GJ, 2005).

According to L.R. Zenkova (2010), the main factors for the effectiveness of depakine chrono include:

* a wide range - idiopathic epilepsy with absences, with myoclonic seizures, cryptogenic epilepsy with generalized seizures, epilepsy with partial seizures, epileptic psychocommunicative disorders (epileptic aphasia, mutism, psychosis);

* efficiency in monotherapy up to 85-90%;

* positive psychocognitive effect;

* the absence or low frequency of cases of intolerance and its predictability;

* positive pharmacodynamic interaction with other

antiepileptic drugs in forced polytherapy;

* stability of therapeutic concentration in plasma and absence of concentration peaks;

* single or double dose;

* good correlation of clinical and neurophysiology effects;

* no effect of paradoxical worsening of seizures.

Carbamazepine is indicated for complex partial seizures. After achieving a therapeutic effect, its dosage can be reduced to the minimum effective level. Begin prescribing carbamazepine with 100-200 mg orally 2-4 times a day, if

necessary, gradually increasing the dose to the usual level of 0.8-1.2 g. per day. It is not recommended to exceed the daily dose of 1.6-2.0 g (Alekseenko Yu.V., 2006).

The standard guidelines for epilepsy treatment are at least 5 years of sustained drug remission. The decision to cancel anticonvulsant therapy is made in the presence of stable clinical and electroencephalographic remission. In post-traumatic epilepsy, the presence of a morphological substrate of the disease makes the risk of recurrence of seizures high in cases of drug withdrawal. Attacks without treatment can resume within 1 year or later (Konovalov A.N., Likhterman L.B., Potapov A.A., 2002; Zenkov L.R., 2010; Karlov V.A., 2010; C hang BS, 2003).

It should be noted that usually post-traumatic epilepsy is difficult to respond to drug therapy (Garga N., 2006), and antiepileptic drugs completely prevent seizures in only 35% of patients with post-traumatic epilepsy (Pitk and nen A., 2006).

A randomized controlled trial revealed the same efficacy of phenotoin, sodium valproate, carbamazepine, phenotropil in the treatment of patients with post-traumatic epilepsy (Agrawal, J., 2006).

In drug-resistant forms of symptomatic epilepsy, surgical treatment is recommended. The purpose of operations used to treat symptomatic epilepsy is to remove the epileptogenic zone. In practice, the term "epileptogenic zone" means the area of persistent epileptic activity, which is the area of seizure generation (Savchenko Yu.N., Savchenko A.Yu., 2007).

M.Ya. Kissin (2011) points to the following forms of surgical interventions used for symptomatic epilepsy:

* open interventions carried out with the help of osteoplastic trepanation of the skull, which allows a differentiated approach in each case to the epileptogenic zone, which can be located at different levels of the brain;

* stereotaxic surgery, which allows you to accurately hit a specific target structure through a burr hole to provide diagnostic studies (subcorticogram) and subsequent therapeutic effects (stimulation or destruction);

* transplantation of embryonic nervous tissue (stem cells) into various brain structures, which makes it possible to improve the electrophysiological characteristics of brain activity, reduce its epileptization, and even out the affective background.

The success of drug therapy and surgery in the treatment of epilepsy is obvious , but in general the problem is still far from being resolved.

Chapter II. MATERIALS AND RESEARCH METHODS

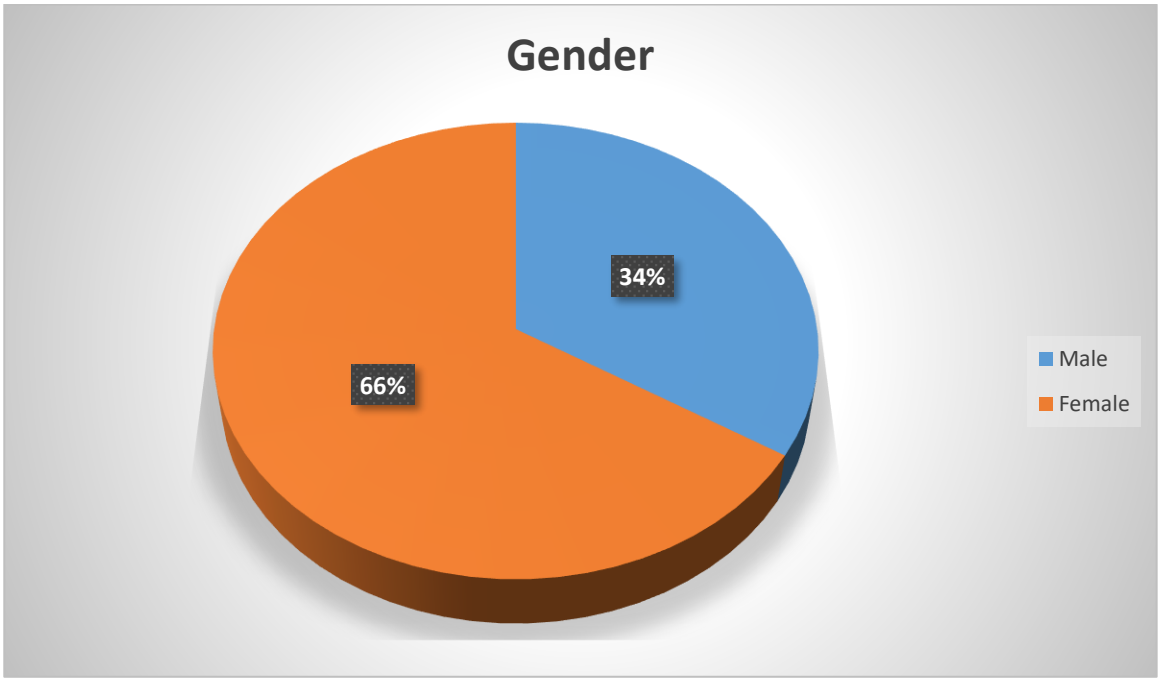
2.1. Characteristics of the examined patients

In the conditions of the department of neurology of the clinic of the Fergana State Medical Institute, we examined 42 patients: patients with post-traumatic epilepsy with a disease experience of 3 years or more from the moment of diagnosis. Among them, 11 women (26.2%) and 31 men (73.8%).

Table 2.1

Distribution of patients in the main group and groups, comparison by sex and age

Age group, male/female	
Age	31.6 ± 1.1 years
Women	33.9%
men	66.1%



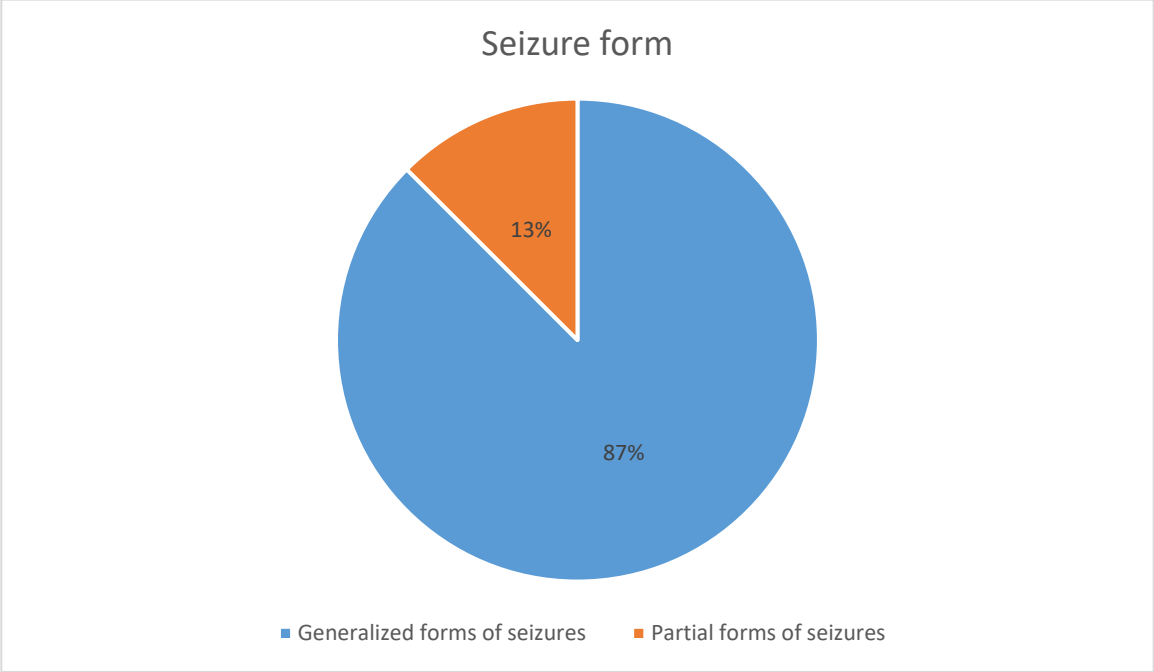
The division of patients according to the types of seizures was carried out in accordance with the International Classification of Epileptic Seizures (1981).

Patients most often encountered a generalized form of epileptic seizures (87.5%), of which 8 patients were after status epilepticus. In 7 patients, a partial form of epileptic seizures (12.5%) was diagnosed.

Table 2.2

The structure of epileptic seizures in the examined patients

Types of epileptic seizures	Patient Ratio
Generalized forms of seizures	87.5%
Partial forms of seizures	12.5%



The distribution of patients by prescription is presented in Table 2.3.

Table 2.3

Disease duration	Occurrence among PTEs
From 3 – yes 5 years	30.3 ± 6.14

6 – 10 years	32.1±6.23
11 – 15 years old	19.7 ± 5.31
Over 15 years	17.9±5.12

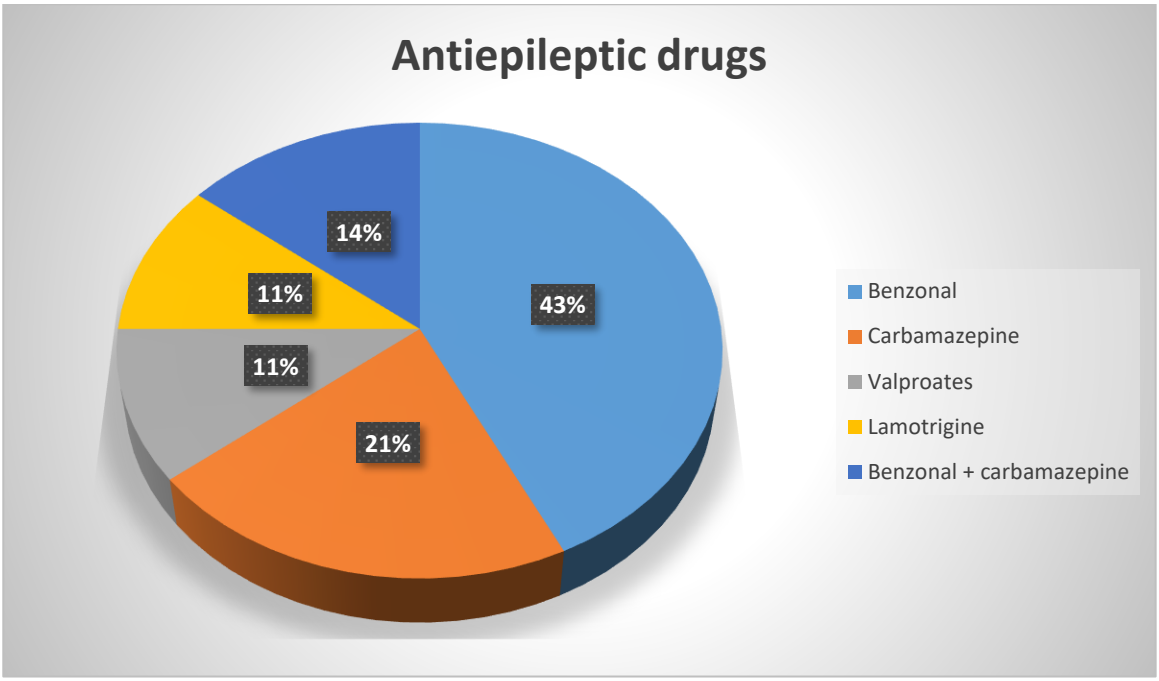
As can be seen from the table, the duration of the disease in patients with PE showed a predominance of patients with a disease duration of up to 10 years, patients with a disease duration of more than 15 years were observed less frequently.

Taking into account the effect of AEDs on the cognitive sphere of patients with epilepsy, a structural analysis of AEDs taken as patients with post-traumatic epilepsy was carried out.

Table 2.4

The structure of anticonvulsants taken in the examined patients

Anti epileptic drug	
Benzonal	42.8%
Carbomazepine	21.4%
Valproates	10.8%
Lamotrigine	10.8%
Benzonal + carbomazepine	14.2%



As shown in Table 2.4, the vast majority (42.8%) of the main group took benzonal tablets at a dose of 100 mg 2-3 times a day as AEDs. In 21.4%, carbamazepine was taken as an AED at a dose of 200 mg 2-3 times a day. Only 6 patients (10.8%) with post-traumatic epilepsy took valproic acid derivatives (depakin, convulex) as AEDs, and 6 patients took lamotrigine.

In 8 cases, patients received combination therapy of benzonal with carbamazepine, which could also have an adverse effect on the cognitive sphere due to increased side effects.

The collection of patients was carried out by filling in the primary material, which included complaints of patients, data on the anamnesis of the disease, taking into account the severity and frequency of TBI, the timeliness and adequacy of conservative and (or) surgical treatment, hereditary burden, and the dynamics of symptoms. All patients underwent a study of the neurological status, a study of the bioelectrical activity of the brain (EEG), and neuroimaging research methods: CT or MRI of the brain. Cognitive functions were examined using neuropsychological tests. The survey was conducted at the beginning of the study and in the dynamics of treatment after 2 months.

2.2. Research methods

2.2.1. Clinical and neurological examination

All groups of patients were subjected to an objective neurological examination according to the generally accepted scheme, which was carried out on the first three days of admission of patients to the hospital.

Clinical and neurological examinations were carried out with a detailed study of the neurological status. They included determining the state of the cranial nerves, the motor sphere (active and passive movements, muscle tone, the presence of paresis and paralysis), the reflex sphere (characteristics of physiological and pathological reflexes), the sensitive sphere (superficial, deep and complex sensitivity), the presence of coordinating disorders .

2.2.2. Methods for assessing cognitive functions.

Mini-mental state examination (MMSE)

To diagnose cognitive impairment, a neuropsychological examination was performed using the following tests and scales: Mini Mental State Examination (MMSE; Folstein M., Pualo J. et al., 1980; Wade, 1992) Mini Mental State Examination (MMSE) Mini-Mental State Examination - MMSE), proposed by Folstein MF and McHugh PR in 1975, evaluates such cognitive functions as the ability of patients to navigate in time and space, perception of information (gnosis), memory, concentration, speech, counting, praxis, executive functions. Only correct answers were counted in the test, and 1 point was awarded for each correct answer. The maximum score for the test is 30. The absence of a cognitive defect was indicated by a result of 28-30 points, 24-27 points were regarded as moderate CR , a result less than or equal to 23 points as dementia.

Frontal dysfunction battery (FAB - frontal assessment battery). The technique has been proposed for the screening of dementia with a predominant lesion of the frontal lobes or subcortical cerebral structures, that is, when the sensitivity of the MMSE may be insufficient.

"Test for memorization of 10 words". The technique is used to study direct short-term, long-term, voluntary and involuntary memorization. The subject is read ten words, selected so that it is difficult to establish any semantic

relationship between them (mountain, needle, rose, cat, clock, wine, coat, book, window, saw). After reading, it is proposed to reproduce the words in any order. Then the words are read again. Normal is the reproduction of 10 words after 4-5 repetitions, with a trained memory after 2 repetitions. After 20-30 min. The subject is asked to reproduce these words in any order.

Spielberger-Khanin Reactive and Personal Anxiety Rating Scale. This scale contains 40 questions (20 - reactive; 20 - personal) and 4 gradations of answers. The answers are marked by the subject himself on the forms and the sum of the points is used to judge the severity of anxiety in the emotional sphere.

2.3. Radiation and neurophysiological diagnostics

Electroencephalography (EEG)

The studies were carried out on an EEG-16S "ME-DIKOR" electroencephalograph. The EEG was recorded after 10 minutes of adaptation of the subject to the conditions of the study. EEG recording was carried out according to the generally accepted method using the J. Jung (1953) scheme when applying electrodes: 4 symmetrical monopolar leads (frontal, central, parietal and occipital), 4 symmetrical bipolar leads (anterior temporal - occipital, anterior temporal - parietal, posterior temporal - frontal, posteriorly - central) - a total of 16 channels.

A background EEG was recorded in a state of relaxed wakefulness. Functional tests were performed with eye opening, photostimulation, a test for 3x minute hyperventilation with EEG registration every minute of hyperventilation and subsequent background EEG recording immediately after the hyperventilation test. The obtained electroencephalograms were subjected to visual analysis with the determination of the main qualitative characteristics of the dominant activity, its regularity, correct zonal distribution, the severity of individual EEG rhythms, the presence and severity of bilateral-synchronous

fluctuations, the presence of foci of pathological activity and pathological phenomena, interhemispheric asymmetry.

The qualitative parameters of the activation reaction were assessed - the degree of desynchronization and extinction of the main activity. The severity of the EEG response to hyperventilation was determined, as well as changes in the qualitative characteristics of the EEG during and after hyperventilation. In addition to the visual, a quantitative analysis of the EEG was also carried out, in which in the EEG section recorded for and 10 sec. the following parameters were determined: 1) the index of the main rhythm, 2) the average amplitude of the main rhythm, 3) the index of slow activity, 4) the index of paroxysmal activity, 5) the amplitude and frequency characteristics of the BSC, 6) the severity of the amplitude-frequency asymmetry of the EEG (B.G .Gafurov, 2005; L.R. Zenkov, M.A. Ronkin, 1991). When assessing the EEG type, we used the classification proposed by E.A. Zhirmunskaya and V.A. Losev (1984).

MRI studies were performed on a SIEMENS MAGNETOM ESSENZA apparatus with a magnetic induction of 1.5 T in standard modes T 1 and T2, in the sagittal and axial planes with a slice thickness of 5 mm.

CT study - used a tomograph ST 8800 (the same company) with a matrix of 256 x 256, image reconstruction in the axial plane and a slice thickness of 10 mm. Contrast enhancement was not performed.

2.4. Statistical processing methods

Statistical processing of the obtained results was carried out on a LENOVO 110 computer using the Microsoft Excel program, taking into account the arithmetic mean (M), the error of the arithmetic mean (m), the linear correlation coefficient (r), the Student-Fisher t-test in the StatSoft software environment , Inc. (2007) when testing the normality of distribution STATISTICA (data analysis software system), version 6. The difference between the compared values was considered significant at $p < 0.05$.

Chapter 3. RESULTS OF CLINICAL AND INSTRUMENTAL STUDIES

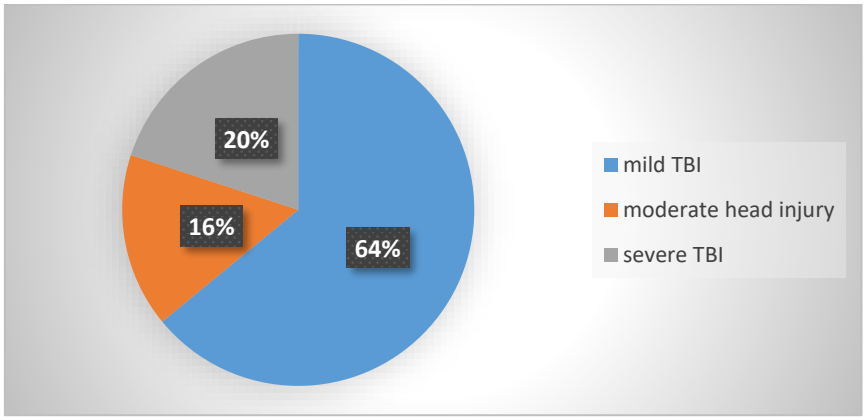
3.1. Clinical and neurological symptoms

42 patients with post-traumatic epilepsy were subjected to an objective clinical, neurological and neuropsychological study .

In patients of the main group, a generalized form of epileptic seizures was most common (87.5%), of which 8 patients were after status epilepticus. In 7 patients, a partial form of epileptic seizures (12.5%) was diagnosed.

At the same time, the timing of the development of post-traumatic epilepsy varied from 2 months to 5 years with the same frequency (in 33.3% after 2 months, in 33.3% after 5 years and in 33.3 % after 4-8 months). In the structure of TBI, which served as the development of subsequent post-traumatic epilepsy, mild TBI prevails, among which in 60.3% of cases there was a repeated TBI, and in 20.7% patients did not seek medical help in a timely manner. In the comparison group, TBI of mild and moderate severity slightly prevailed.

Rice. 1. Structure of TBI in patients with post-traumatic epilepsy



Among patients with post-traumatic epilepsy selected for our study, 30.3% received TBI in childhood. Of these, in 4 patients (7.1%), when testing for a battery of frontal dysfunction, severe dementia was detected (all took benzonal tablets as AED), the duration of the disease at the time of the

examination was from 8 to 36 years. In 1 child after severe TBI, cognitive impairment arose and progressed to the development of post-traumatic epilepsy.

The most frequent complaints in patients with post-traumatic epilepsy were complaints of emotional lability, headaches, and irritability.

Table 3.1

The structure of subjective complaints of patients in the surveyed groups.

Complaints of patients	Occurrence
Headache	85.7+4.67
Dizziness	80.3+5.31
Memory impairment	64.2+6.4
Memory disorders	67.8+6.24
Fast fatiguability	62.5+6.46
Irritability	83.9+4.91
Emotional lability	91+3.82

A comparative analysis of the neurological status in patients revealed a significant predominance of focal symptoms in patients with post-traumatic epilepsy.

Table 3.2

The structure of neurological symptoms in patients with post-traumatic epilepsy

Symptoms	Occurrence
Soreness of the epicranial muscles	85.7 ± 4.67
nystagmus	91±3.82

Strabismus	3.5±2.45
Weak convergence	5.32±2.99
Soreness of Valle points	85.7±4.67
Central paresis of 7 and 12 pairs	76.7±5.64
dysarthria	3.5±2.45
Hemiparesis	3.5±2.45
Hemihypoesthesia	1.78±1.76
Anisoreflexia	87.5±4.67
hyperreflexia	12.5±4.41
Potological reflexes	3.5±2.45
Reflexes of oral automatism	55.3±6.64
Instability in the Romberg position	91±3.82
Intention tremor	75±5.78

As can be seen from Table 3.2, the most common symptomatology in patients with post-traumatic epilepsy was cerebellar-discoordinating syndrome, manifested as instability in the Romberg position (91%), in second place in terms of frequency of occurrence in 85.7% was pain in the points of Valle, in 76, 7% had central paresis of the VII and XII pairs, less often there was an installation nystagmus of a unilateral and bilateral nature (28.5% and 23.2%, respectively).

In addition, in patients of the main group, a picture of hemiparesis was more often observed (in 5 patients who had a severe head injury followed by a craniotomy.

3.2. The state of the general mental status of the examined

When testing patients according to the MMSE test, a significant predominance of mild dementia in patients with post-traumatic epilepsy was revealed (37.5%)

Table 3.3

Indicators of the mental status of the examined patients

	MMSE	Forehead Test Battery
Post-traumatic epilepsy	22.2±0.58	12.7±0.38
	0.05	0.05

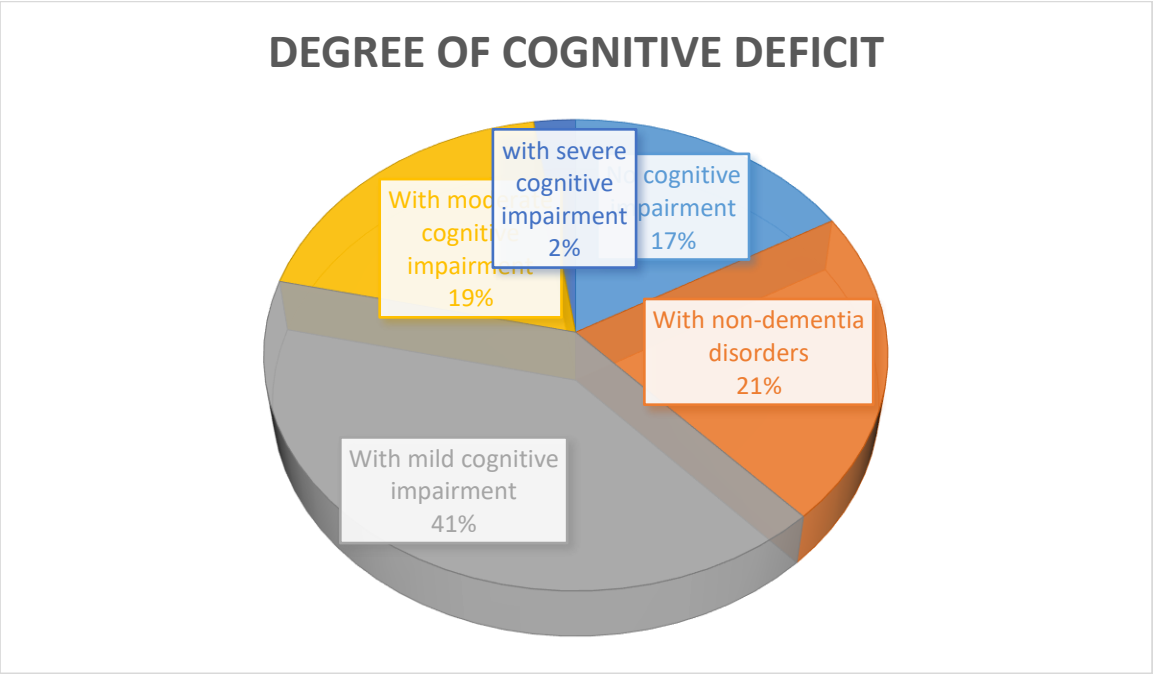
As can be seen from Table 3.3, mean MMSE scores in patients with post-traumatic epilepsy corresponded to mild cognitive impairment (22.2 points), and mean scores for the frontal dysfunction battery corresponded to moderate dementia.

In 6 patients with post-traumatic epilepsy (10.7%), no cognitive impairment was detected according to the MMSE test. Also, in patients of the main group, cases of moderate dementia were more often noted according to the MMSE test.

However, given the insensitivity of the MMSE test to disturbances in the functioning of the frontal lobes, an additional test was performed on the frontal dysfunction battery, which made it possible to more reliably detect severe dementia (Table 3.4.).

Among patients with post-traumatic epilepsy with a partial form (7 people), there were no cases of severe dementia: 20% showed no cognitive impairment, 20% showed signs of non-dementia disorders, 40% had mild cognitive impairment, 20% had moderate dementia with a disease duration of 10 years with frequent attacks, in which benzonal was used as an AED.

The structure of cognitive impairments according to the MMSE test in patients



Degree of cognitive deficit	Frequency of occurrence
No cognitive impairment	16.66%
With non-dementia disorders	21.42%
With light KN	40.46%
With moderate KN	19.04%
With heavy KN	2.38%

All cases of moderate and severe dementia occurred in patients with a generalized form of post-traumatic epilepsy.

Table 3.5

Indicators of auditory-speech memory

10 Word Memory Test Averages (number of words)			
	Immediately after	After 10 minutes	In 20 minutes

	learning		
PTE	5.6±0.2	3.8±0.24	2.9±0.3
	p <0.001	p <0.001	p <0.001

A detailed analysis of cognitive impairment in patients revealed the predominance of involuntary memory impairment in its auditory-speech modality in patients with post-traumatic epilepsy (88.6%), while they occurred both in patients with severe and moderate dementia, and in patients with non-dementia disorders.

According to the test for memorization of 10 words in the main group, patients who did not have cognitive impairments reproduced from 5 to 8 words 20 minutes after memorization. Patients with non-dementia cognitive impairment reproduced from 4 to 8 words. Patients with post-traumatic epilepsy with mild cognitive impairment reproduced from 4 to 6 words after 20 minutes (naming words that did not exist or were close in meaning). Patients with moderate dementia - from 0 to 2 words (naming words that did not exist or are close in meaning). Patients with severe dementia could not reproduce a single word from the very beginning of memorization.

In second place in terms of frequency of occurrence were disorders of concentration when performing the task to read the word backwards (MMSE test), as well as when counting in the mind, which were 3.5 times more common in patients.

Table 3.6

Indicators of auditory-speech memory in patients with various degrees of cognitive impairment

Research methods	10 Word Memory Test Averages (number of words)

Patient groups	Immediately after learning	After 10 minutes	In 20 minutes
Without KN	7.0±0.63	6.2±0.65	4.8 ± 0.79
With non-demented KN	6.3±0.28	5±0.26	4.2±0.24
With light KN	5.4±0.27	2.9±0.24	2.3±0.41
With moderate KN	4.5±0.43	2.5±0.27	1.2±0.25
With heavy KN	3	1	0

Tests for constructive praxis in patients with post-traumatic epilepsy with severe and moderate dementia revealed violations in drawing geometric shapes according to the standard. At the same time, a complete discrepancy between the figures was noted in patients examined on days 2-3 after status epilepticus (7%).

Table 3.7

The Structure of Cognitive Disorders in Patients of the Three Study Groups

cognitive functions	Frequency of cognitive impairment
1) orientation in time	14.2±4.6
2) orientation in place	7.1±3.4
3)orientation in self	0
4) involuntary memory	87.5±4.4
5) understanding of speech and complex logical and grammatical structures	28.5±6.0

6) expressive speech	16.0±4.8
7) dynamic praxis	46.4± 6.6
8) constructive praxis	55.3±6.6
9) reading	28.5±6.0
10) letter	35.7±6.4
11 attention span	80.3±5.3

Clinical example 1.

Patient D., 43 years old, diagnosed with post-traumatic epilepsy, generalized form, condition after status epilepticus. From the anamnesis: in 1994 he received a CBI, a concussion of the brain, was not treated in a hospital. Periodically received outpatient treatment. Over the past 5-6 years, headaches bothered me. On the day of admission to the clinic, the first generalized tonic-clonic seizure developed, which subsequently assumed a status character. In a state of epileptic status, he was taken to the clinic. Examined on the 3rd day after status epilepticus. According to the battery of frontal dysfunction - a severe degree of dementia. Makes no complaints. Disoriented in time and place, reduced criticism, excited. Answers questions briefly, reluctantly. When drawing geometric figures according to the standard, there is a complete discrepancy and distortion of the picture. Wrote slurred words.

When asked to answer the questions of the Spielberger-Khanin test, he began to agree in all answers, then crumpled the questionnaire sheet, after which the testing was terminated. Orientation in his own personality is preserved, correctly calls his name, surname, age. On the EEG (against the background of lamitor 150 mg/ day and benzonal 300 mg/day) - gross changes in the

bioelectrical activity of the brain. A focus of epileptic activity emanating from the mid-stem structures of the brain.

Violations of counting operations and constructive praxis, according to the literature, indicate the defeat of the parietal and parietal-temporal region of the right hemisphere. However, characteristic disorders of constructive praxis are also observed in cases of damage to the right hemisphere, which makes it impossible to evaluate such phenomena without analyzing the entire set of data from neuropsychological studies.

Disorders of dynamic praxis (according to the battery of frontal dysfunction) in patients with status epilepticus were characterized by erroneous execution of hand movements in all three attempts, but a high level of claims (overestimation of one's own capabilities). The EEG in these cases recorded subclinical epileptiform activity (15%). According to literary sources, it is subclinical epileptiform activity that often aggravates the cognitive and emotional sphere of patients.

Patients with post-traumatic epilepsy, who did not have pronounced cognitive impairment, realistically assessed their abilities in the interictal period and stated the fact that it was impossible to perform tests.

Writing disorders were associated with a decrease in motivation, only 4.8% of patients had signs of agrammatism. In cases of severe dementia, perseveration phenomena (rewriting of the task from the questionnaire) were noted. In patients with traumatic brain disease, writing disorders were associated with the presence of severe neurological symptoms (paresis).

Reading disorders were noted in patients who had traumatic brain injury in childhood and adolescence (30.1%) and were associated with the pedagogical neglect of patients. Classical alexia associated with impaired reading center was not found.

Patients experienced significant difficulties due to impaired understanding of speech and complex logical and grammatical structures in the absence of signs of sensory aphasia. However, these violations were detected only with a

thorough examination. Patients did not actively complain about difficulties in understanding complex information. Patients with severe dementia were unable to independently read the questions of the Spielberger-Khanin test, which he proposed to assess the level of anxiety, and answer them. Already when they read out the questions, it was revealed that they gave contradictory answers, and in patients examined after status epilepticus, as well as in patients with subclinical epileptiform activity on the EEG (15%), echolalia phenomena and the choice of all four answers to the question were noted. Therefore, it was decided to regard the results of the Spielberger - Xanine test in patients with severe dementia as unreliable.

In the study of expressive speech, violations of the repetition of syllables and phrases were revealed, and dialogic and spontaneous speech remained intact.

Disturbances in orientation in time and place were found only in patients with severe dementia, which were characterized by incorrect naming of the real date, month, year, season and country in which the patient is located.

Orientation in one's own personality has always been preserved, even in patients who have undergone status epilepticus.

Anxiety in patients with epilepsy acts as a secondary mental disorder due to the reaction of the individual to information about the presence of a significant disease. Anxiety disorders and depression in such patients are associated with psychogenic and external environmental influences, which significantly affect the quality of life.

Anxiety disorders were highly variable. Patients with post-traumatic epilepsy without cognitive impairment were characterized by an average degree of reactive anxiety, which corresponds to the normative indicators of healthy individuals and is regarded as an adequate assessment of reality. And indicators of personal anxiety corresponded to its severe degree. As the cognitive deficit worsened in patients with post-traumatic epilepsy, there was a tendency to reduce the degree of reactive and personal anxiety. So, in patients with a

moderate degree of dementia, a mild degree of reactive anxiety and a severe degree of personal anxiety, but less expressed in quantitative terms, were revealed.

Table 3.8

Indicators of reactive and personal anxiety of the examined patients

	Reactive	Personal
PTE	28.3±1.9	48.3±2.3
R	>0.05	>0.05

Taking into account gender (gender) differences in the emotional sphere, we studied the severity of reactive and personal anxiety separately in males and females in each of the studied groups.

Table 3.9

Indicators of anxiety depending on the degree of cognitive deficit

	Reactive	Personal
Without KN	38.2 ± 4.82	56.5 ± 4.76
With non-demented KN	28.9 ± 2.71	49.3 ± 2.31
With light KN	30.8± 2.92	53.6 ± 2.51
With moderate KN	19.6± 5.13*	34.5 ± 8.4*
With heavy KN	0	0

Note: An asterisk marks the significance in relation to the data of patients without cognitive impairment (* - $p < 0.05$; ** - $p < 0.01$).

Indicators of reactive anxiety (RT) in males in both examined groups were identical, and mild RT was detected most often (up to 30 points according to the Spielberger-Khanin test). In the women of the main group, there was a

predominance of RT of moderate severity, while in the comparison group there was a more even distribution of RT indicators with a slight predominance of RT of moderate severity (from 30 to 45 points).

Indicators of personal anxiety (PT) in men of the main group corresponded to moderate and severe equally. And in the comparison group, mild LT was noted in 3.8% of cases. In women with post-traumatic epilepsy, severe LT was detected in 100% of cases (≥46 points according to the Spielberger-Khanin test), while in the comparison group, severe LT occurred only in 84.2% of cases. Thus, cognitive disorders in patients with post-traumatic epilepsy are more pronounced and are characterized by impaired memory, attention, understanding of speech and complex logical and grammatical structures, anxiety disorders and depend mainly on the frequency of seizures and the AED taken and, accordingly, the bioelectrical activity of the cerebral cortex.

An analysis of the degree of cognitive impairment depending on the duration of the pathological process revealed certain patterns (Table 3.10). Thus, in patients with post-traumatic epilepsy, cognitive impairment did not depend on the duration of the disease and corresponded to severe dementia according to the battery of frontal tests, while mild cognitive impairment was determined by the MMSE test.

Table 3.10

Mean indicators of the degree of cognitive impairment depending on the duration of the disease.

PTE duration	MMSE test	Forehead Test Battery
3 to 5 years	22.3±1.24	13.2 ± 0.66
6-10 years old	22.2±0.84	12.7±0.64
11-15 years old	21.4±1.21	12.2±0.89

15 years or more	23.5±1.32	12.9±0.99

The indicators of auditory-speech memory according to the test for memorizing 10 words in patients with post-traumatic epilepsy with a disease duration of up to 5 years averaged 5.9 words immediately after memorization, which is worse than the normative indicators of healthy people, and after 10 and 20 minutes they were characterized by a decrease in reproducible words by 1, 7 and 0.7 words, respectively (Table 3.11). Every 5 years of the disease, the indicators of auditory-speech memory were characterized by a progressive deterioration, which is apparently associated with the involvement of mediobasal structures in the epileptic process, which normally provide the processes of memory consolidation.

Table 3.11

Average indicators of auditory-speech memory according to the test for memorization of 10 words, depending on the duration of the disease with post-traumatic epilepticus.

PTE duration	Immediately after learning	After 10 minutes	In 20 minutes
3 to 5 years	5.9±0.38	4.3±0.42	3.7±0.57
6-10 years old	6.1±0.31	3.8±0.40	2.9±0.43
11-15 years old	5.2±0.25	3.3±0.29	2.6±0.49
15 years or more	4.4±0.47	2.9±0.46	2.1±0.56

A comparative study of anxiety disorders in patients of the three study groups, depending on the duration of the disease, revealed the presence of mild reactive anxiety and severe personality anxiety in patients with post-traumatic epilepsy and idiopathic epilepsy during the first 5 years of the disease, while

patients with post-traumatic encephalopathy had moderate reactive anxiety. severity and severe degree of personal anxiety, quantitatively exceeding this indicator in patients of the main group and patients with idiopathic epilepsy.

Table 3.12

Mean rates of anxiety disorders depending on the duration of the disease.

PTE duration	reactive anxiety	Personal anxiety
3 to 5 years	25.9±3.3	13.2 ± 0.66
6-10 years old	22.2±0.84	12.7±0.64
11-15 years old	21.4±1.21	12.2±0.89
15 years or more	23.5±1.32	12.9±0.99

A comparative study of anxiety disorders in patients of the three study groups, depending on the duration of the disease, revealed the presence of mild reactive anxiety and severe personality anxiety in patients with post-traumatic epilepsy and idiopathic epilepsy during the first 5 years of the disease, while patients with post-traumatic encephalopathy had moderate reactive anxiety. severity and severe degree of personal anxiety, quantitatively exceeding this indicator in patients of the main group and patients with idiopathic epilepsy.

As the disease progressed, patients of the main group tended to worsen anxiety disorders in the form of reactive anxiety of moderate severity and severe personal anxiety (increased by an average of 12.2 points).

3.3. Features of sensorimotor reactions and attention

It is known that when studying sensorimotor reactions according to the Schulte tables, healthy individuals spend an average of 40-50 seconds (Bleikher

V.M. Pathopsychological diagnostics, 1987), and sometimes there is even an acceleration in the rate of sensorimotor reactions in subsequent tables. If the search is conducted unevenly, then this is either a sign of increased exhaustion, or belated development. Of great importance in this case is the "curve of exhaustion of attention", built graphically on the basis of the results of the experiment, since it can objectively reflect the nature of asthenia.

In the study of attention switching in the main group, in 23.2% of cases, patients could not perform a single action according to the modified Gorbov table; 76.8% completed only 1 and 2 actions (separate search for black numbers in ascending order and red numbers in descending order). Only 7.1% (4 patients) were able to complete the last third task. In the group of patients with idiopathic epilepsy, 17.5% of patients were unable to complete all three tasks, and only 5 patients were able to complete the third task. In the comparison group with post-traumatic encephalopathy, 88.5% of patients completed the first two tasks, and 11.5% could not complete any task.

In patients with post-traumatic epilepsy, who did not have any cognitive impairments according to the MMSE test and the frontal dysfunction battery, a high initial level was revealed (mean 76.2 seconds) and there was a tendency to reduce the time spent searching for numbers in subsequent tables (59 seconds for the last table). table), which indicates an acceleration in the rate of sensorimotor reactions. A similar picture was observed in patients with idiopathic epilepsy (72 seconds for the first and 65 seconds for the last table).

In the study of attention switching in this group of patients, positive results were obtained in 42.8%, their attention switching coefficient was 0.63 seconds (the rest of the patients did not cope with the task at its various stages). The initial indicators of the rate of sensorimotor reactions in patients with post-traumatic epilepsy with non-dementic cognitive impairment were 9.7 seconds better than in patients without cognitive impairment, however, from the second Schulte table, they developed irregular signs of increased attention exhaustion and an “exhaustion curve”. 81.8% of patients could not cope with the third task

in the study of attention switching and made mistakes after completing from a quarter to a half of the task. The average attention span was 1.07 seconds.

In the comparison group, in patients with non-demented cognitive impairment, the initial indicators of the rate of sensorimotor reactions did not differ much from the normative indicators of healthy people, however, the "exhaustion curve" corresponded to the hyposthenic form of asthenia with a steady decrease in indicators, which indicates an unfavorable prognosis and is a sign of disease progression .

5.2% of patients could not perform any action according to the modified Gorbov table; 89.4% completed only 1 and 2 actions (separate search for black numbers in ascending order and red numbers in descending order); 5.2% (1 patient) performed all 3 actions and the attention switching coefficient was 0.68 seconds.

Indicators of the rate of sensorimotor reactions in patients of the main group with signs of mild dementia were 30.9 seconds worse than in patients of the comparison group, however, they were characterized by a hypersthenic form of the "exhaustion curve". From the very beginning, 31.2% of patients (5 people) could not perform tests for switching attention from the very beginning, the rest of the subjects in this group performed only 1 and 2 actions. Due to their failure to perform 3 actions, the coefficient of switching attention could not be determined.

In the comparison group with mild cognitive impairment, the initial indicators of the rate of sensorimotor reactions were identical to those of patients in the main group. However, already from the second table, a significant deterioration in performance was noted, which, apparently, is associated with increased exhaustion.

Switching attention in this group was low. From the very beginning, 6.2% of patients failed to complete the task. The same number of patients coped with only the first action. The initial indicators for the first action of both groups were approximately the same (107 and 109.6 seconds), however, patients with post-

traumatic encephalopathy were characterized by an improvement in indicators in the second action, while patients with post-traumatic epilepsy showed a negative trend. For one patient from both groups with signs of moderate dementia, they could not complete the tasks according to any of the tables presented.

In patients with post-traumatic epilepsy, the rates of sensorimotor reactions and switching of attention are much higher than in patients with post-traumatic encephalopathy, which is apparently due to the predominance of patients with focal brain lesions and severe neurological symptoms in the latter.

The average indicators of attention in patients of the three studied groups with a disease duration of up to 5 years were characterized by the presence of an attention exhaustion curve of the "wrong" type, for which a pattern of increasing and decreasing the time spent searching for numbers in each Schulte table is typical.

However, the indicators of attention in patients with post-traumatic epilepsy were worse compared to those of the two comparison groups. As the disease progressed, patients with post-traumatic epilepsy were characterized by a tendency to a quantitative and qualitative deterioration in the indicators of attention and sensorimotor reactions, expressed in the transformation of the "attention exhaustion curve" into a "hyposthenic" type, which is characterized by a progressive increase in the time spent on finding numbers in each subsequent Schulte table. The same trend was observed in patients with idiopathic epilepsy.

In patients with post-traumatic encephalopathy, attention indicators also deteriorated over time, however, the "attention exhaustion curve" did not take on a "hyposthenic" character, but corresponded to an "incorrect" type.

When analyzing the switchability and selectivity of attention according to the modified Gorbov table in patients of the main group, a tendency to an increase in the time spent on the task of finding red numbers in descending order was determined. And the third task (alternately showing black numbers in ascending order, and red numbers in descending order) was completed by one

patient in the group with a disease duration of up to 5 years, from 6 to 10 years. The rest of the patients were more likely to make mistakes at the beginning of the third task, and these indicators were annulled.

For patients with post-traumatic encephalopathy, as well as patients with idiopathic epilepsy with a disease duration of up to 10 years, there was a tendency to reduce the time spent on completing the 2nd task according to the modified Gorbov table, which indicates the preservation of the development of attention characteristic of healthy people. Patients with post-traumatic encephalopathy were also characterized by an improvement in performance over time.

In patients with idiopathic epilepsy, after 10 years of the disease, there was a tendency to worsen indicators of selectivity and switching of attention, however, in quantitative terms, they exceeded similar indicators of patients in the main group.

3.4. Features of cognitive disorders in patients with post-traumatic epilepsy depending on the antiepileptic drug taken

In the general structure of patients with post-traumatic epilepsy selected for our study, patients (42.8%) took benzonal tablets as an AED, patients (21.4%) - carbamazepine, patients took a combination of benzonal with carbamazepine (14.2%), patients were taking valproic acid derivatives (10.7%) and 6 patients were taking lamotrigine (10.7%).

Since our task was not to change the AED, we limited ourselves to a comparative assessment of individual indicators of the cognitive status of our patients (Table 3.14).

Comparative characteristics of the degree of cognitive deficit depending on the anticonvulsant drug taken showed that the worst indicators of cognitive impairment were observed in patients who took benzonal tablets (12.7 and 22.6 points) and the combination of benzonal with carbamazepine (11.6 and 20.4 points) . Higher rates were obtained for patients taking valproate (15.5 and 25.7 points) and lamotrigine (14.7 and 24.8 points).

Hearing-speech memory in the 10-word memorization test was directly proportional to the AED taken and the degree of cognitive deficit. As can be seen from Table 3.15, the lowest rates of voluntary memory were observed in patients taking Benzonal, while 1 patient with severe dementia could not reproduce a single word, both from the very beginning of memorization and 10 and 20 minutes after memorization.

Table 3.14

Average indicators of the general mental status of patients with post-traumatic epilepsy, depending on the AED taken

	Benzonal + carbam	Benzonal	Carbamazepine	Valproates	Lamotrigine
MMSE test	20.4±1.3	22.6±0.83	23.3±0.84*	25.7±0.86*	24.8±0.84*
Forehead Test Battery	11.6±0.68	12.7±0.55	13.5±0.57*	15.5±0.58*	14.7±0.74*

Note: * - significance between indicators ($p < 0.05$).

Table 3.15

Average indicators of auditory-speech memory in patients with post-traumatic epilepsy, depending on the AED taken

	Benzonal + carbamazepine	Benzonal	Carbamazepine	Valproates	Lamotrigine
Immediately after learning	5.38±0.43	5.13±0.35	6.25±0.34	7.17±0.48	7.0±0.54

After 10 minutes	3.63±0.50	3.38±0.36	4.08±0.49	5.83±0.41*	5.9±0.48*
In 20 minutes	2.88±0.57	2.71±0.37	3.25±0.60	6.17±0.57*	6.3±0.67*

Note: * - significance between indicators (p <0.05).

Two patients from this group and one patient from the carbamazepine group could not reproduce a single word 20 minutes after memorization. The best indicators of arbitrary memory were found in patients of the group of lamotrigine and valproic acid derivatives. A progressive decrease in the number of reproducible words was noted in the benzonal group, the combination of benzonal with carbamazepine, and in the carbamazepine group, which indicates impaired memory consolidation processes and fixing information in long-term memory.

Patients from the group of lamotrigine and valproic acid derivatives were characterized by a qualitatively and quantitatively stable tendency to fix information in long-term memory, expressed in the reproduction of more words 20 minutes after memorization than 10 minutes after memorization, while the number of reproduced words corresponded to standard indicators . healthy people (more than 6 words).

The average indicators of attention and the rate of sensorimotor reactions in patients taking the combination of benzonal with carbamazepine turned out to be the worst and were characterized by the formation of a "hypersthenic type" of the attention exhaustion curve. Tests for switching attention according to the Gorbov table could be performed only by one patient from this group, spending 131.8 seconds on the first task and 143.6 seconds on the second task. The patient did not cope with the third task (alternately finding red numbers in descending order and black numbers in ascending order).

For patients taking benzonal and carbamazepine as monotherapy, the curve of attention exhaustion of the "wrong" type was characteristic. In quantitative terms, patients in the carbamazepine group outnumbered those in the benzonal group by an average of 15 seconds. One patient from the benzonal group and one patient from the carbamazepine group were able to perform tests on attention switching, while the initial indicators of attention of the patient from the carbamazepine group were 23 seconds better than the patient taking benzonal. When performing the third task, the difference was 61 seconds in favor of the patient from the carbamazepine group.

Note : * - reliability between indicators ($p < 0.05$) .

A feature of the properties of the attention of patients in the lamotrigine and valproic acid group was the relative compliance of the indicators with the normative indicators of healthy people, while in patients taking valproic acid derivatives, there was a clear tendency to develop attention, manifested in the acceleration of the pace of sensorimotor reactions as tasks were completed according to the Schulte tables.

Comparative analysis of the level of anxiety in patients of different groups revealed an average level of reactive anxiety in patients of the benzonal group and the combination of benzonal with carbamazepine. In the remaining groups of patients, the level of reactive anxiety corresponded to a mild degree of its severity with the lowest score in the carbamazepine group. All patients were characterized by severe personal anxiety, most pronounced in patients taking benzonal.

3.5. Functional state of the cerebral cortex according to electroencephalography data

Changes in the brain in epilepsy are reflected in the characteristics of the bio-electrical activity of the brain, assessed using electroencephalography (EEG). Electroencephalography is one of the main methods for objective testing of the functions of the nervous system. Being an almost ideal method for

directly displaying the functioning of the CNS, EEG solves the problems of diagnosing not only organic, but also functional brain lesions (3, 34, 68). As is known, the EEG depends on the mechanisms that determine the level of functional activity of the entire brain - the limbic-reticular complex, non-specific systems of the brain. The features of these systems are their median location in the brain, their diffuse and symmetrical connection with the cortex. The EEG changes significantly under the influence of the pathological process, and its changes depend on the severity and nature of the brain damage. According to the pattern of changes in the rhythms of the biopotentials of the cortex, it is possible to identify focal disorders caused by a local pathological process.

Bioelectrical activity in 29.5% of patients with PTE was not significantly changed. The background EEG was characterized by a regular alpha rhythm, with the correct regional distribution and frequency . amplitude modulation. In patients with idiopathic epilepsy, a regular alpha rhythm with a correct regional distribution was observed in 23.4% of cases, while in patients with post-traumatic encephalopathy in 56.7% of cases.

Studies of the bioelectric activity of the brain in our patients showed that the most pronounced disorganization of the alpha rhythm was observed in the group of patients with idiopathic epilepsy and PTE. In 80.4% of patients, a pronounced violation of the regularity, correct regional distribution and frequency-amplitude modulations of alpha activity and a significant increase in the slow activity index (IMA) were revealed . In 45.9% of cases, alpha activity was absent on the EEG. In these cases, fast activity dominated in the range of low-frequency beta rhythm (14-20 Hz) or irregular slow activity in the range of theta or delta oscillations (Table 3.16). In patients with idiopathic epilepsy, in 70% of cases, there was a pronounced disorganization of the alpha rhythm , and in 53.3% it was absent. At the same time, in patients with PTE, the percentage of patients with impaired organization of the dominant rhythm was significantly lower than in the comparison groups.

The index of slow activity in the group of patients with PE exceeded those in epilepsy, but the results were not statistically significant. The index of slow activity was significantly lower in patients with post-traumatic encephalopathy. Pathological activity in the form of "outbursts" of fluctuations of biopotentials of various kinds was observed more often in post-traumatic epilepsy (28.4%). Significantly less in idiopathic epilepsy and PE (19.4% and 1.5%), respectively).

Table 3.16

Alpha-rhythm indices in examined patients

	Alpha Rhythm Features				
	Absent	Frequency		Regional distribution	
		Regular	Irregular	Saved	Smoothed out
PTE	45.9±6.4	19.7+5.1	34.4±6.1	9.8±3.8	44.3±6.4

They were expressed on the EEG more often bilaterally and more often at the same time in all, less often in the anterior parts of the brain. "Flashes" had a duration of 1-3 seconds. and consisted of beta - oscillations of low frequency, waves in the range of theta - and delta - frequencies. In most cases, the "flashes" were clear with an amplitude noticeably enhanced compared to the background activity. Similar shifts on the EEG indicate a pronounced dysfunction of the median structures.

Epileptic activity in the form of sharp waves, peaks, "acute-slow wave" complexes was recorded in 55.7% of patients with PE and in 63.3% of patients with epilepsy. It should be noted that in 11.3% of patients with post-traumatic encephalopathy, epileptic activity was recorded on encephalograms, although there was no indication of the presence of epileptic seizures in the anamnesis.

Paroxysmal activity in 89.6% of cases came from the mid-stem structures, in 10.4% there was a focus of epileptic activity in one or another area of the brain.

Table 3.17

Indicators of epileptic activity

	Availability	Absence
Patients with PTE	55.7	44.3

In order to systematize the obtained data, we analyzed the EEG of the examined patients using the classification of E.A. Zhirmunskaya and V.Losev (61). As is known, the authors of this classification distinguish 20 EEG groups with different qualitative and quantitative gradations from normal to gross pathology. The totality of individual groups with about 5 types of EEG: Type I - organized in time and space; II type - synchronous, monorhythmic; III type - d asynchronous; IV type - disorganized with a predominance of alpha activity; Type V - disorganized with a predominance of slow activity. The results of the EEG classification of our patients are presented in Table 3.18.

Table 3.18

Distribution by EEG types

	EEG types				
	I	II	III	IV	V
PTE	6.6±3.2	18.0±4.9	9.8±3.8	29.5±5.8	36.1±6.1

From the presented data, it can be seen that the most pronounced changes were observed in patients with idiopathic epilepsy and PTE. Thus, the disorganized type with a predominance of high-amplitude slow waves (type V) was recorded in 43.3% and 36.1%, respectively, which was significantly higher

than in patients with TBM (18.9%). At the same time, a significant increase in the percentage of hypersynchronous EEG type was noted (23.7% and 18.0%, respectively), which in some cases indicates an increased convulsive readiness of the brain. At the same time, normal or close to normal (types I and III) EEG pattern was significantly higher in patients with TBM. Next, we compared the state of the bioelectrical activity of the brain depending on the state of the cognitive sphere of patients with PE.

Table 3.19

Comparative characteristics of EEG types and cognitive deficits

	EEG types				
	I	II	III	IV	V
Without KN	38.5±13.5	-	30.8±12.8	23.1±11.7	7.7±7.4
Light KN	25.0±10.8	6.3±6.1	37.5±12.1	18.8±9.8	12.5±8.3
Moderate KN	-	26.7±11.4	6.7±6.4	40.0±12.6	26.7±11.4
Heavy KN	-	41.2±11.9	-	23.5±10.3	35.3±11.6

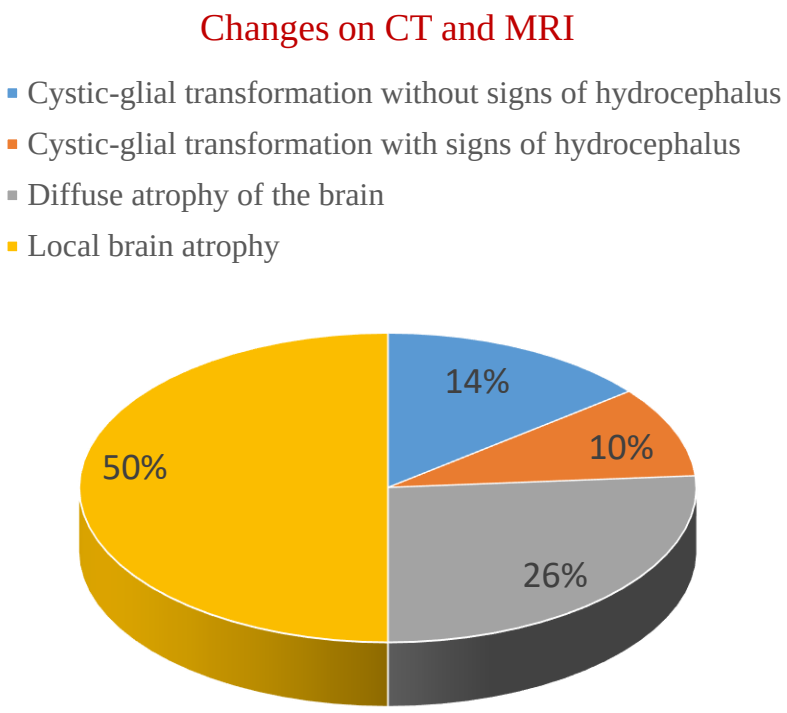
Thus, the obtained results indicate a gross disorganization of the EEG in patients with PE, and the degree of impairment increases with the degree of cognitive deficit. At the same time, epileptic activity is recorded in patients with TBGM, indicating subclinical epileptic activity, which must be taken into account when determining the plan of drug therapy.

3.6 Dynamics of indicators of the neuroimaging research method

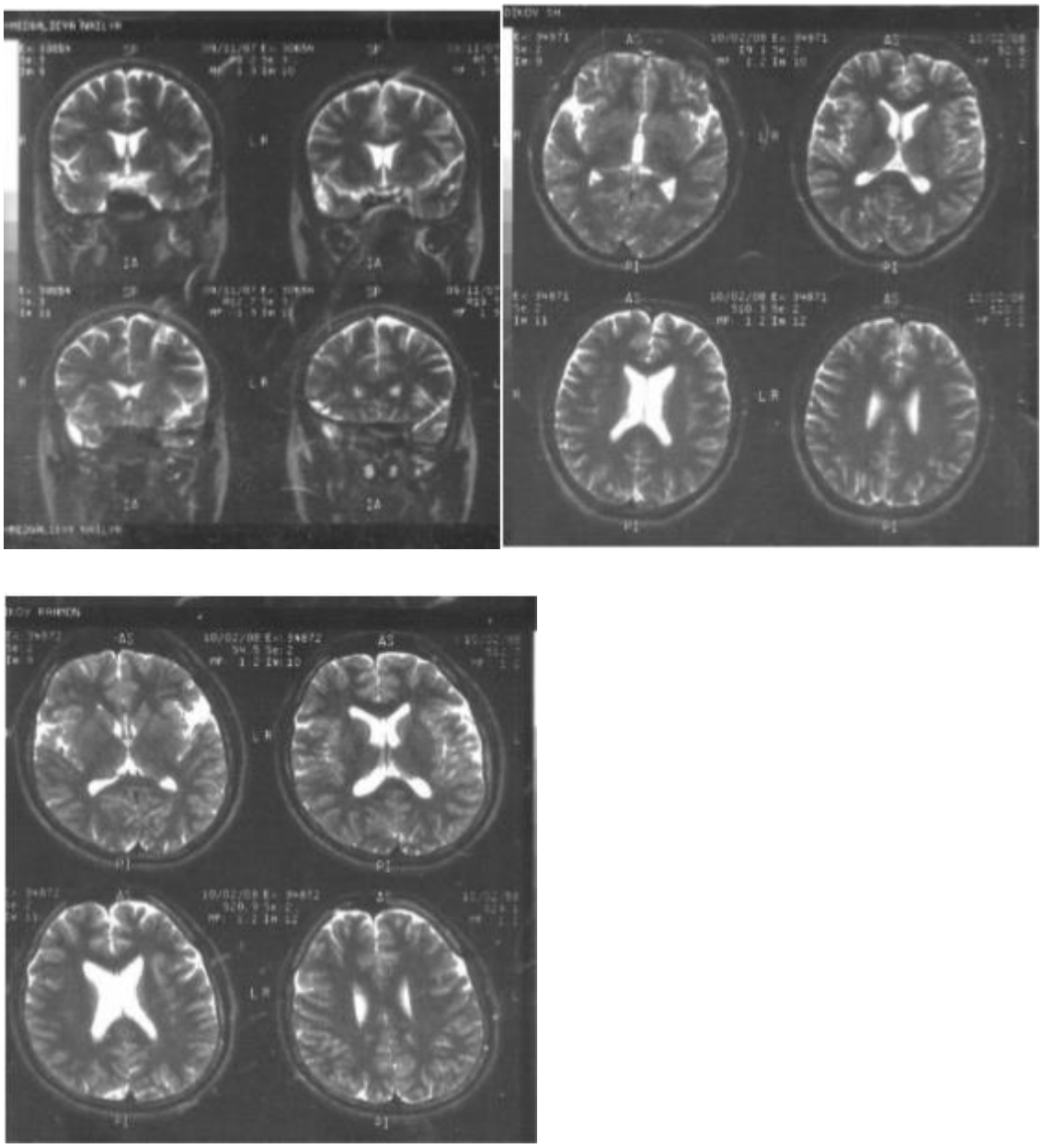
The main substrate of post-traumatic brain injuries is focal damage to neurons, which is morphologically manifested by hypoxic and sclerotic changes

in the brain tissue, the formation of collagenous, hyal and cerebrospinal fluid cysts. All studied patients underwent an MRI study. MRI study was characterized by morphological changes: cystic-glial transformation without hydrocephalus: - 6 (15%); cystic-glial transformation with hydrocephalus: - 4 (10%); diffuse atrophy of the brain: - 10 (25%); local brain atrophy: - 20 (50%) (tab. 3.20).

Table 3.20



In patients with post-traumatic epilepsy, focal morpho-destructive changes are statistically more represented, which can serve as a diagnostic, prognostic indicator, and was used to evaluate the criteria for optimizing treatment.



Rice. 3.1. MRI of the brain in patients with post-traumatic epilepsy

3.7. DYNAMICS OF COGNITIVE DISORDERS ON THE BACKGROUND OF COMPLEX THERAPY

The issue of therapy of cognitive disorders in epilepsy has always encountered difficulties due to the impossibility of nootropic therapy due to its proseizure activity. The synthesis of a new group of nootropic drugs, phenylpiracetam, which is part of the drug Phenotropil, gave hope for a solution to this issue.

The results of numerous studies have shown that this drug has the ability to suppress epileptic activity through various mechanisms and have a nootropic effect [4, 6, 41, 57].

Obtaining the latest information about the nature of memory disorders involving neurobiochemical changes at various levels: neuronal, synaptic, membrane, cellular, molecular, made it possible to purposefully approach the search for means of correcting these disorders, resulting in a number of new substances that have significant nootropic activity and original mechanisms of action.

Patients with post-traumatic epilepsy were taking the drug phenotropil at a dosage of 100 mg 2 times a day for 2 months.

The results of the dynamic observation of patients revealed a positive trend in patients taking fenotropil. Despite the slightly low initial indicators of auditory speech , patients showed a significant increase in the number of word reproduction immediately after memorizing words by 1.1 and 1.2 words, respectively (Table 4.1). At the same time, a further study showed that in the group of patients who took phenotropil, there was a significantly more pronounced increase in the stability of attention and retention of information in memory, and, accordingly, the reproduction of more words after 10 and 20 minutes. Thus, one can think that phenotropil has a more positive effect on the processes of memory and attention than pantocalcin.

Table 3.21

Indicators of auditory-speech memory in patients with post-traumatic epilepsy before and after taking phenotropil preparations.

Average number of words on a memory test 10 words	Phenotropil use	
	Before treatment	After treatment

Immediately after learning	5.8±0.22	6.5±0.21*
After 10 minutes	3.8±0.3	5.1±0.26**
In 20 minutes	2.9±0.33	4.3±0.28**

Note: the asterisk indicates the reliability in relation to the original data. * - $p < 0.05$; ** - $p < 0.01$

When analyzing the indicators of concentration of attention in patients with post-traumatic epilepsy who took phenotropil, a trend was revealed to a significant improvement in indicators in the form of a decrease in the time spent on finding numbers according to the first three Schulte tables (on average by 33% for all tables) and a quantitative approximation to the normative indicators of healthy people. people (72.9 sec.). However, the attention exhaustion curve corresponded to the “hyposthenic” type, which is characterized by a gradual increase in the time spent on completing a task according to each subsequent Schulte table, which is a sign of the progression of cognitive disorders in and posttraumatic epilepsy.

A similar trend also took place when performing tasks according to the modified Gorbov table, designed to determine the selectivity and switchability of attention. In the dynamics of treatment with phenotropil, 89.3% of patients completed the first and second tasks compared to the initial data (78.6% of patients). The third and last task was completed by 7 patients, which accounted for 25%.

The initial indicators of attention in patients of the main group who took pantocalcin corresponded to the "hyposthenic" type of the attention exhaustion curve. In the dynamics of treatment, there was a quantitative improvement in indicators (by 26%), however, the degree of shortening of the time was much

smaller compared to the previous group and statistically unreliable in all four tables (averaging 81.8 seconds).

A gradual and uneven decrease in indicators was noted, as a result of which a “hypersthenic” type of attention exhaustion curve was formed. The results of the study of selectivity and shifting of attention according to the modified Gorbov table revealed a decrease in the time spent on each subsequent task. 85.7% of patients coped with the first two tasks, however, the time spent on tasks significantly exceeded the indicators of patients receiving phenotropil. Only three patients of the group coped with the third task on the switching of attention.

The use of Phenotropil preparations in the complex therapy of cognitive disorders in patients of the main group and the comparison group gave positive results, characterized by an improvement not only in individual indicators of memory and attention, but also in a general improvement in cognitive status, determined by the results of a battery of frontal tests and the MMSE test (Table 4.2) .

Table 3.22

Indicators of the dynamics of cognitive disorders in patients with post-traumatic epilepsy on the background of complex therapy

Tests (in points)	Phenotropil use	
	Before treatment	After treatment
Frontal lobe battery	12.3±0.54	13.4±0.57
MMSE	21.5±0.80	23.7±0.71*

The results of a dynamic neuropsychological examination in the phenotropil group revealed a significantly positive trend in the form of an increase in the level of cognitive status by 2.2 points only according to the MMSE test (p <0.05). The complex use of phenotropil in patients of both groups revealed a positive trend also in relation to anxiety disorders (Table 4.3).

In the group of patients taking phenotropil, the average indicators of reactive anxiety initially corresponded to its mild degree and slightly decreased after treatment, while the indicators of personal anxiety increased slightly.

Table 3.23

Indicators of anxiety before and after taking Phenotropil preparations in patients with post-traumatic epilepsy

Anxiety indicators	The use of Phenotropil	
	Before treatment	After treatment
Reactive	26.6±2.5	24.5±1.02 *
Personal	46.8±3.16	51.8±1.9 *

Note: an asterisk indicates the reliability in relation to the original data. * - p <0.05.

Analysis of the EEG structure at the beginning of the study showed a significant predominance of pathological EEG types and rare conditionally normal types. The results of EEG observation 2 months after the course of therapy showed a significant decrease in the frequency of desynchronous EEG types.

Table 4.4

Comparative characteristics of EEG - pictures in patients with post-traumatic epilepsy in the dynamics of the study

EEG types	The use of Phenotropil	
	Before treatment	After treatment
I type	10.7±5.8	35.7±9.1*
II type	14.3±6.6	3.6±3.5
III type	14.3±6.6	21.4±7.8
I V type	21.4±7.8	10.7±5.8
V type	39.3±9.2	28.6±8.5

Note: * - reliability in relation to the original data ($p < 0.05$)

For the completeness of the qualitative characteristics of the EEG of patients, we evaluated its frequency spectrum. The mid-frequency characteristics of the main EEG rhythm at the beginning of the study in the group of patients treated with fenotropil was 9.82±0.21 Hz, and at the end of the study - 10.64±0.12 Hz.

Epileptiform changes on the EEG were assessed according to their International Classification with registration of peaks, sharp waves, peak-wave complexes. These data are presented in Table 4.5.

Table 3.24

Characteristics of epileptic activity on the EEG in the dynamics of therapy

epileptic activity	The use of Phenotropil	
	Before treatment (%)	After treatment (%)

Availability	57.1±9.4	39.3±9.2 *
Absence	42.9±9.4	60.7±9.2 *

Note: * - reliability in relation to the original data ($p < 0.05$)

When analyzing pathological changes on the EEG at the beginning of the study, their rather high level attracted attention. Nevertheless, the analysis of epileptic activity on the EEG at the end of the study showed that a statistically significant decrease in epileptic activity was observed in patients receiving fenotropil.

Thus, the results of our study revealed the positive effect of phenotropil on the cognitive sphere of patients with post-traumatic epilepsy. The effect of phenotropil was more pronounced in relation to the indicators of auditory memory and attention. This drug improved the state of the bioelectrical activity of the brain, characterized by a decrease in EEG disorganization.

The use of phenotropil in complex therapy in patients with post-traumatic epilepsy allows, by improving cognitive functions, to maintain their social adaptation longer, and therefore improve the quality of life.

CONCLUSION

Considering the problem of a mnestic-intellectual defect in epilepsy, it should be emphasized that it is not characterized by a specific phenomenology that would allow it to be distinguished from a defect resulting from another organic pathology [52]. The presence and nature of a cognitive defect is influenced by a large number of factors related both directly to the disease itself (the form of epilepsy, the type and frequency of seizures, the presence of morphological changes in the study of the brain, their severity, the age of onset of the disease and the age of the patient), and to the pharmacotherapy of epilepsy. It is well known that patients are forced to take antiepileptic drugs almost throughout their lives. At the same time, patients taking several drugs, or with a high level of them in the blood plasma, are highly likely to develop neurotoxic effects, which also manifests itself in a decrease in cognitive functions [52, 55, 107, 136]. This forces clinicians to carefully weigh the severity of the side effects of antiepileptic drugs when choosing them and give preference to drugs with the lowest risk of developing neurotoxicity. It is also not clear under the influence of which particular antiepileptic drugs cognitive

impairments increase, which, ultimately, should aggravate the overall severity of the mnestic-intellectual defect.

The problem of cognitive impairment currently attracts the attention of doctors of various specialties - neurologists, psychiatrists, gerontologists, neuropsychologists. Degenerative diseases of the central nervous system, especially epilepsy, are among the causes of persistent, sometimes irreversible cognitive disorders. For a long time, the main attention was focused on severe cognitive disorders reaching the stage of dementia, when the quality of life of patients and those around them is seriously impaired.

Unfortunately, attempts at therapeutic intervention at this stage of the disease do not yet give the effect that would suit both doctors and patients in due measure. At the same time, early initiation of therapy at the stage of "pre-dementia" disorders can slow down the progression of the disease and delay the appearance of a pronounced cognitive deficit [1, 5].

The issue of therapy of cognitive disorders in epilepsy has always encountered difficulties due to the proconvulsant activity of many nootropic drugs.

A comprehensive examination of patients, seizure control, rehabilitation programs allow patients to adapt to the changed living conditions. However, a true assessment of the effectiveness of implemented medical and rehabilitation measures can be obtained by taking into account the parameters of the quality of life of a patient who finds himself in connection with the disease in changed living conditions [13, 14].

In the conditions of the department of neurology of the clinic of the Fergana State Medical Institute, we examined 42 patients: patients with post-traumatic epilepsy. The main group consisted of patients with post-traumatic epilepsy: women (34%) and men (66%) aged 16 to 47 years, whose average age was 31.6±1.1 years. Of these, patients with a generalized form of epilepsy (87.5%) and 7 patients with a partial form (12.5%).

In order to improve the cognitive functions of patients with post-traumatic epilepsy, Phenotropil preparations were used for 2 months. After 2 months, the above methods of research were again carried out by the patients , and the results were recorded in the primary material. A dynamic assessment of the cognitive sphere of patients in two comparison groups was also carried out.

When testing patients according to the MMSE test, a significant predominance of mild dementia in patients with post-traumatic epilepsy (37.5%) was revealed.

The average values of the MMSE test in patients with post-traumatic epilepsy corresponded to mild cognitive impairment (22.2 points), and the average values for the frontal dysfunction battery corresponded to moderate dementia and averaged 12.8 points.

Considering the insensitivity of the MMSE test to disorders of the functioning of the frontal lobes, an additional test was carried out on the frontal dysfunction battery, which made it possible to more reliably detect severe dementia.

Among patients with post-traumatic epilepsy with a partial form, there were no cases of severe dementia: in 20% no cases were detected
no cognitive impairment, 20% showed signs of non-dementia disorders, 40% had mild cognitive impairment, 20% had moderate dementia with a disease duration of 10 years with frequent attacks, in which benzonal was used as AED.

All other cases of moderate and severe dementia occurred in patients with a generalized form of post-traumatic epilepsy. In the group of patients with post-traumatic epilepsy selected for our study, 30.1% received TBI in childhood. Of these, in 4 patients (7.5%), when testing for a battery of frontal dysfunction, severe dementia was detected (all of them took benzonal tablets as AED), the duration of the disease at the time of the examination was from 8 to 36 years. In both study groups, a small proportion of patients with severe TBI was revealed: 25% of patients in the main group and patients with post-traumatic encephalopathy in the comparison group (21.1%). Patients with idiopathic

epilepsy did not have a history of TBI. A detailed analysis of cognitive impairment in patients of the two study groups revealed the predominance of involuntary memory impairment in its auditory-speech modality in patients with post-traumatic epilepsy (88.6%). At the same time, they occurred both in patients with severe and moderate dementia, and in patients with non-dementia disorders.

The course of epilepsy is accompanied by a steady weakening of memory (Bleikher V.M.). Initially, a violation of arbitrary reproduction is detected - concentration of attention on the reproduction of a word in memory leads to a deterioration in the ability to reproduce. At later stages, retention and memory disorders are found. Such dynamics of memory disorders in epilepsy was noted by O. Bumke, N. Shipkovensky. A similar sequence of memory impairment is also characteristic of cerebral atherosclerosis without gross focal pathology. This gives grounds to believe that the weakening of voluntary reproduction, followed by a deterioration in retention and memorization, is not a feature of any particular disease, but is inherent in diseases with slowly progressive mnestic disorders.

Mild violations in the form of literal substitutions occur only when presentation of long and unfamiliar words like "Gibraltar", "electroencephalogram". These symptoms occur in 70% of patients with left temporal and 30% of patients with right temporal irritation of the epileptic focus and do not occur in patients with lesions of the fronto-central region.

Of interest is a certain correspondence between the severity and depth of memory disorders and general progressive oligophasia in epilepsy. The less memory disorders are expressed, the less pronounced is the general progressive oligophasia, and, conversely, more pronounced oligophasic manifestations correspond to deeper memory disorders.

In second place in terms of frequency of occurrence were disorders of concentration when performing the task to read the word backwards (MMSE

test), as well as when counting in the mind, which were 3.5 times more common in patients of the main group.

Tests for constructive praxis in patients with post-traumatic epilepsy with severe and moderate dementia revealed violations drawing geometric shapes according to the standard. At the same time, a complete discrepancy between the figures was noted in patients examined on days 2-3 after status epilepticus (7%).

Violations of counting operations and constructive praxis, according to the literature, indicate damage to the parietal and parietal-temporal region of the right hemisphere. However, characteristic disorders of constructive praxis are also observed in lesions of the right hemisphere, which makes it impossible to evaluate such phenomena without analyzing the totality of data from neuropsychological studies [6, 16, 131]. Disorders of dynamic praxis (according to the battery of frontal dysfunction) in patients with status epilepticus were characterized by erroneous execution of hand movements in all three attempts, but a high level of claims (overestimation of one's own capabilities). The EEG in these cases recorded subclinical epileptiform activity (15%). According to literature sources, it is subclinical epileptiform activity that often aggravates the cognitive and emotional sphere of patients [105, 132, 142].

Writing disorders were associated with a decrease in motivation, only 4.8% of patients had signs of agrammatism. In cases of severe dementia, the phenomena of rewriting the task from the questionnaire were noted. In patients with traumatic brain disease, writing disorders were associated with the presence of severe neurological symptoms (paresis).

Reading disorders were noted in patients who had traumatic brain injury in childhood and adolescence (30.1%) and were associated with the pedagogical neglect of patients. Classical alexia associated with impaired reading center was not found.

Patients experienced significant difficulties due to impaired understanding of speech and complex logical and grammatical structures in the absence of

signs of sensory aphasia. However, these violations were detected only with a thorough examination. Patients did not actively complain about difficulties in understanding complex information. Patients with severe dementia were unable to independently read the questions of the Spielberger-Khanin test, which he proposed to assess the level of anxiety, and answer them. Already when they read out the questions, it was revealed that they gave contradictory answers, and in patients examined after status epilepticus, as well as in patients with subclinical epileptiform activity on the EEG (15%), echolalia phenomena and the choice of all four answers to the question were noted. Therefore, it was decided to regard the results of the Spielberger-Khanin test in patients with severe dementia as unreliable.

In the study of expressive speech, violations of the repetition of syllables and phrases were revealed, and dialogic and spontaneous speech remained intact.

Disturbances in orientation in time and place were found only in patients with severe dementia, which were characterized by incorrect naming of the real date, month, year, season and country in which the patient is located.

Orientation in one's own personality has always been preserved, even in patients who have undergone status epilepticus.

Anxiety in patients with epilepsy acts as a secondary mental disorder due to the reaction of the individual to information about the presence of a significant disease. Anxiety disorders were highly variable across the 3 study groups. Patients with post-traumatic epilepsy without cognitive impairment were characterized by an average degree of reactive anxiety, which corresponds to the normative indicators of healthy individuals and is regarded as an adequate assessment of reality. And indicators of personal anxiety corresponded to its severe degree. As the cognitive deficit worsened in patients with post-traumatic epilepsy, there was a tendency to reduce the degree of reactive and personal anxiety. So, in patients with a moderate degree of dementia, a mild degree of

reactive anxiety and a severe degree of personal anxiety, but less expressed in quantitative terms, were revealed.

Patients with post-traumatic encephalopathy had a completely different picture of anxiety disorders. Patients without cognitive disorders were characterized by a mild degree of reactive anxiety and a moderate degree of personal anxiety. As the cognitive deficit increased, there was a tendency to increase anxiety. For example, patients of this group with moderate dementia had an average degree of reactive anxiety and a severe degree of personal anxiety.

Patients with idiopathic epilepsy were also characterized by an aggravation of the degree of anxiety as the cognitive deficit increased.

Taking into account sexual (gender) differences in the emotional sphere, we

studied the severity of reactive and personal anxiety separately in males and females in each of the study groups. Indicators of reactive anxiety (RT) in males in both examined groups were identical, and mild RT was detected most often (up to 30 points according to the Spielberger-Khanin test). In the women of the main group, there was a predominance of RT of moderate severity, while in the comparison group there was a more even distribution of RT indicators with a slight predominance of RT of moderate severity (from 30 to 45 points).

Indicators of personal anxiety (PT) in men of the main group corresponded to moderate and severe equally. And in the comparison group, mild LT was noted in 3.8% of cases. In women with post-traumatic epilepsy, severe LT was detected in 100% of cases (≥46 points according to the Spielberger-Khanin test), while in the comparison group, severe LT occurred only in 84.2% of cases.

According to D.M. Andreiko, in patients with frequent seizures in situational anxiety, the overall indicator of anxiety with emotional discomfort was significantly higher, and the phobic component prevailed in personal anxiety in comparison with patients with rare seizures. The relationship between the asthenic component of situational anxiety in persons engaged in mental and

other types of labor has been traced. Potential anxiety in persons engaged in mental activity, even in cases where data were obtained on a significant predominance of the asthenic component in persons engaged in mental work, in the form of rapid fatigue, difficulty in concentrating, lethargy in comparison with patients not engaged in mental activity, in dominated by a phobic component and an anxious assessment of perspective.

In patients with post-traumatic encephalopathy, cognitive impairments were not so pronounced and corresponded to mild cognitive impairments according to the battery of frontal tests, and according to the MMSE test, to non-dementia cognitive impairments. In addition, patients in this group were characterized by a tendency to improve cognitive status after 10-15 years from the moment of injury. This fact is confirmed by literary sources (Gilyarovsky V.A.), indicating the development of pseudo-organic dementia resulting from TBI. After the symptoms caused by the functional components of a traumatic brain injury have passed, the organic core of dementia remains, and the course of the disease becomes more stable for a long time.

In patients with idiopathic epilepsy, the picture of the dynamics of cognitive disorders depending on the duration of the disease was characterized by the presence of mild impairments during the first 5 years of the disease, both according to the MMSE test and the battery of frontal dysfunction, however, in this group there was a tendency to a pronounced progression of cognitive disorders from 5 to 15 years of disease, when cognitive impairment reaches a severe degree of dementia, more clearly detected by testing the frontal dysfunction battery , while moderate dementia was detected by the MMSE test.

Comparative characteristics of the degree of cognitive deficit depending on the anticonvulsant drug taken showed that the worst indicators of cognitive impairment were observed in patients who took benzonal tablets (11.4 and 21.6 points) and the combination of benzonal with carbamazepine (11.6 and 20.4 points) . Higher rates were obtained for patients taking valproate (15.5 and 25.6 points) and lamotrigine (14.7 and 24.8 points) ($p < 0.05$).

The lowest rates of voluntary memory were observed in patients taking Benzonal, while 1 patient with severe dementia could not reproduce a single word, both from the very beginning of memorization and 10 and 20 minutes after memorization. The best indicators of arbitrary memory were found in patients of the group of lamotrigine and valproic acid derivatives. A progressive decrease in the number of reproducible words was noted in the benzonal group, the combination of benzonal with carbamazepine, and in the carbamazepine group, which indicates impaired memory consolidation processes and fixing information in long-term memory.

Patients from the group of lamotrigine and valproic acid derivatives were characterized by a qualitatively and quantitatively stable tendency to fix information in long-term memory, expressed in the reproduction of more words 20 minutes after memorization than 10 minutes after memorization, while the number of reproduced words corresponded to standard indicators. healthy people (more than 6 words).

Thus, in post-traumatic epilepsy, more pronounced cognitive impairment is observed, compared with idiopathic epilepsy and post-traumatic encephalopathy. Memory and attention disorders prevail in the structure of cognitive disorders . The severity of cognitive impairment depends on the severity of TBI, the frequency of seizures, and the antiepileptic therapy being taken.

The results of our study revealed the role and significance of timely neuropsychological diagnosis of cognitive disorders in patients with post-traumatic epilepsy, as well as the positive effect of Phenotropil on the cognitive sphere of patients with post-traumatic epilepsy.

CONCLUSIONS

1. Post-traumatic epilepsy is dominated by moderate and severe cognitive impairment with a predominant impairment of memory and concentration. As the disease progresses, the apracto-agnostic syndrome joins, a violation of the understanding of complex logical and grammatical structures.

2. Psycho-emotional status in patients with post-traumatic epilepsy is characterized by a decrease in reactive anxiety and an increase in personal anxiety as the disease progresses.

3. The use of Phenotropil has a positive effect on the cognitive sphere of patients, regresses the development of reactive anxiety disorders, leads to a decrease in epileptic activity and normalization of the neurophysiological state

PRACTICAL RECOMMENDATIONS

1 . For a more reliable diagnosis of the degree of cognitive disorders in post-traumatic epilepsy, it is advisable to use several complementary methods.

2. In the complex therapy of cognitive disorders in patients with epilepsy with a predominance of anxiety disorders, and with impaired attention and memory, Phenotropil should be used.

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APPLICATION

Annex 1

Methods for assessing cognitive functions Mini-mental state examination (MMSE)

1. Orientation in time. The maximum score (5) is given if the patient independently and correctly names the date, day of the week, month, year and season. Each mistake or lack of answer reduces the score by 1 point.

2. Orientation in place . The question is asked: "Where are we?" If the patient does not fully answer, additional questions are asked. The patient must name the country, region, city, institution in which the examination takes place, floor. Each mistake or lack of answer reduces the score by 1 point.

3. Perception. Instructions are given: "Repeat and try to remember three words: apple, table, coin." Words should be pronounced as clearly as possible at a speed of one word per second. For each correctly reproduced word, 1 point is awarded. The words should be presented as much as necessary for the subject to repeat them correctly, however, only the first repetition is scored.

4. Concentration of attention. They are asked to subtract sequentially from 100 to 7. Five subtractions are enough. Each mistake reduces the score by 1 point. If the patient is unable to complete this task, he is asked to pronounce the word "earth" backwards. Each mistake reduces the score by 1 point.

5. Memory. The patient is asked to remember the words that were memorized in paragraph 3. Each correctly named word is estimated at 1 point.

6. Speech functions. They show a pen and ask: “What is this?”, similarly - a watch. Each correct answer is worth 1 point.

Ask the patient to repeat the phrase " No if, no but." Correct repetition is worth 1 point.

A command is given verbally, which provides for the sequential execution of the above three actions. Each action is worth 1 point.

A written instruction is given (for example, "Close your eyes"), the patient is asked to read it and follow it. The instructions must be written in sufficiently large block letters on a clean sheet of paper.

The patient must independently write a meaningful and grammatically complete sentence.

The patient is given a sample (two crossed rectangles with equal angles), which he must redraw on clean, unlined paper. If spatial distortions or non-connection of lines occur during redrawing, the execution of the command is considered incorrect. This does not take into account the distortion of figures due to tremor.

Interpretation of results: The result of the test is obtained by summing the results for each of the items. The maximum score in this test is 30 points, which corresponds to the highest cognitive abilities. The lower the test result, the more pronounced the cognitive deficit.

- 28-30 points - no cognitive impairment
- 24 - 27 points - non-dementia cognitive impairment
- 20-23 points - mild cognitive impairment
- 11-19 points - moderate dementia
- 0-10 points - severe dementia

Appendix 2

Frontal dysfunction battery (FAB - frontalassessmentbattery)

The technique has been proposed for the screening of dementia with a predominant lesion of the frontal lobes or subcortical cerebral structures, that is, when the sensitivity of the MMSE may be insufficient.

1. Conceptualization. The patient is asked: "What do an apple and a pear have in common?" The answer that contains a categorical generalization ("It's a fruit") is considered correct. If the patient finds it difficult or gives a different answer, he is told the correct answer. Then they ask: “What is in common between a coat and a jacket?” ... “What is in common between a table and a chair?”. Each

categorical generalization is worth 1 point. The maximum score in this subtest is 3, the minimum is 0.

2. Fluency of speech. They are asked to close their eyes and for a minute say words starting with the letter “s”. In this case, proper names are not counted. Result: more than 9 words per minute - 3 points, from 7 to 9 - 2 points, from 4 to 6 - 1 point, less than 4 - 0 points.

3. Dynamic praxis. The patient is invited to repeat a series of three movements after the doctor with one hand: fist (placed horizontally, parallel to the surface of the table) - rib (the brush is placed vertically on the medial edge) - palm (the brush is placed horizontally, palm down). At the first presentation of the series, the patient only follows the doctor, at the second presentation, he repeats the movements of the doctor, and finally, he does the next two series on his own. When self-fulfillment tips to the patient are unacceptable. Result: correct execution of three series of movements - 3 points, two series - 2 points, one series (together with the doctor) - 1 point.

4. Simple reaction of choice. The instruction is given: “Now I will check your attention. We will tap out the rhythm. If I hit once. You must hit twice in a row. If I strike twice in a row, you must strike only once.” The following rhythm is tapped out: 1-1-2-1-2-2-2-1-1-2. Evaluation of the result: correct execution - 3 points, no more than 2 errors - 2 points, many errors - 1 point, complete copying of the doctor's rhythm - 0 points.

5. Complicated choice reaction. The instruction is given: “Now if I strike once, you must do nothing. If I strike twice in a row, you must strike only once.” The rhythm is tapped: 1-1-2-1-2-2-2-1-1-2. Evaluation of the result is similar to paragraph 4.

6. Study of grasping reflexes. The patient sits, he is asked to put his hands on his knees with palms up and check the grasping reflex. The absence of a grasping reflex is estimated at 3 points. If the patient asks if he should grab, a score of 2 is given. If the patient grabs, he is instructed not to, and the grasping reflex is

retested. If the reflex is absent during the re-examination, 1 is assigned, otherwise - 0 points.

Thus, the test result can vary from 0 to 18; while 18 points correspond to the highest cognitive abilities.

Appendix 3

"Test for memorization of 10 words".

The technique is used to study direct short-term, long-term, voluntary and involuntary memorization. The subject is read ten words, selected so that it is difficult to establish any semantic relationship between them (mountain, needle, rose, cat, clock, wine, coat, book, window, saw). After reading, it is proposed to reproduce the words in any order. Then the words are read again. Normal is the reproduction of 10 words after 4-5 repetitions, with a trained memory after 2 repetitions. After 20-30 min. The subject is asked to reproduce these words in any order.

The following indicators are noted: 1) the number of words reproduced; 2) quantitative dynamics of the reproduced words (arbitrary memorization curve). According to the results of this test, the following conclusions about the characteristics of the subject's memory are possible:

1. Direct memorization is not impaired - in cases where the subject immediately after reading ten words to him reproduces at least 7 words in four or five attempts.
2. Direct memorization is impaired - in cases where the subject immediately after reading ten words to him reproduces less than 7 words. The smaller the number of words the subject manages to reproduce, the more pronounced violations of direct memorization are recognized.

Appendix 4

Reactive and Personal Anxiety Rating Scale

Spielberger-Khanin

This scale contains 40 questions (20 - reactive; 20 - personal) and 4 gradations of answers. The answers are marked by the subject himself on the forms and the sum of the points is used to judge the severity of anxiety in the emotional sphere.

Instruction.

1. “Read carefully each of the following sentences and circle the appropriate numbers to the right, depending on how you feel at the moment. Don't think about the questions for a long time, because there are no right or wrong answers.
2. “Now, in exactly the same way, read carefully each of the sentences below and circle the appropriate number to the right, depending on how you usually feel. Like last time, don't think too long about the answers."

Reactive anxiety data processing: first calculate the sum of the underlined digits for items (sentences) 3, 4, 6, 7, 9, 12, 13, 14, 17, 18. Then get the sum of the underlined digits for items 1, 2, 5, 8, 10, I, 15, 16, 19, 20. Subtract the second from the first sum and add 35 to the result.

Processing the results of personal anxiety: first of all, the underlined numbers in the answers should be summarized: 2, 3, 4, 5, 8, 9, 11, 12.14, 15, 17, 18, 20; then subtract from the result the sum of the underlined digits of the answers: 1, 6, 7, 10, 13, 16, 19; add 35 to the resulting difference.

Data interpretation (same for reactive and personal anxiety):

• up to 30 points - low anxiety;
• 31 -45 points - moderate anxiety;
• 46 points or more - high anxiety.

Appendix 4

Electroencephalography (EEG)

The studies were carried out on an EEG-16S " ME DIKOR" electroencephalograph. EEG registration was performed after 10 minutes of adaptation of the subject to the conditions of the study. EEG recording was carried out according to the generally accepted method using the J. Jung (1953)

scheme when applying electrodes: 4 symmetrical monopolar leads (frontal, central, parietal and occipital), 4 symmetrical bipolar leads (anterior temporal - occipital, anterior temporal - parietal, posterior temporal - frontal, posteriorly - central) - a total of 16 channels.

A background EEG was recorded in a state of relaxed wakefulness. Functional tests were performed with eye opening, photostimulation, a test for 3x minute hyperventilation with EEG registration every minute of hyperventilation and subsequent background EEG recording immediately after the hyperventilation test. The obtained electroencephalograms were subjected to visual analysis with the determination of the main qualitative characteristics of the dominant activity, its regularity, correct zonal distribution, the severity of individual EEG rhythms, the presence and severity of bilateral-synchronous fluctuations, the presence of foci of pathological activity and pathological phenomena, interhemispheric asymmetry.

The qualitative parameters of the activation reaction were assessed - the degree of desynchronization and extinction of the main activity. The severity of the EEG response to hyperventilation was determined, as well as changes in the qualitative characteristics of the EEG during and after hyperventilation. In addition to the visual, a quantitative analysis of the EEG was also carried out, in which in the EEG section recorded for and 10 sec. the following parameters were determined: 1) the index of the main rhythm, 2) the average amplitude of the main rhythm, 3) the index of slow activity, 4) the index of paroxysmal activity, 5) the amplitude and frequency characteristics of the BSC, 6) the severity of the amplitude-frequency asymmetry of the EEG (B.G .Gafurov, 2005; L.R. Zenkov, M.A. Ronkin, 1991). When assessing the EEG type, we used the classification proposed by E.A. Zhirmunskaya and V.A. Losev (1984).

MRI studies were performed on the MAGNETOM OPENviva SIEMENS apparatus with a magnetic induction of 0.5 T in standard modes T 1 and T2, in the sagittal and axial planes with a slice thickness of 5 mm.