**MINISTRY OF HEALTH CARE OF THE REPUBLIC OF UZBEKISTAN**

**TASHKENT STATE INSTITUTE OF DENTISTRY**

# COGNITIVE IMPAIRMENT IN STROKE PATIENTS WITH PANIC ATTACKS.

**(Monograph)**

**Tashkent-2025**

**THE MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN**

**TASHKENT STATE INSTITUTE OF DENTISTRY**

**«ТАСДИҚЛАЙМАН»**

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**«____»_____ 2025 й.**

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Cognitive disorders accompanied by panic attacks in stroke patients. Monograph/ Sh.Sh.Khikmatullaeva.

This monograph describes the main cognitive impairments in stroke patients, studies the etiological and pathogenetic mechanisms, clinical and differential diagnosis of stroke in patients with ischemic stroke in patients with hypersensitivity to cholecystitis and hypersensitivity to insulin.

The monograph is intended for a wide range of neurologists, clinical residents, students, masters, and doctoral candidates.

The monograph was reviewed by the Internal Diseases Committee of the Tashkent State Dental Institute (---) and the Scientific Council of the Tashkent State Stomatological Institute (---).

**Scientific secretary, Ph.D., professor** **A.A. Yuldashev**

CONTENTS

## LIST OF ABBREVIATIONS

II–ischemic stroke

AB–arterial pressure

VBH–vertebro-bazillyar pool

KH–carotid pool

UuA–general the artery sleep

TuA–external sleep arteriesc

IUA–sleep, inner arteriesc

OMa–previous brain arteriesc

O'Ma–middle cerebral arteryc

OMa–of the spinal cord arteriesc

MWFK–tserebrovaskulya'likla r sick

HS–quality of life

Kb–cognitive disorders

VX–panic attacks

CNS–central nervous system

## INTRODUCTION

"Stroke remains one of the leading causes of mortality and long-term disability in developed countries. According to the European Stroke Organization, severe disability occurs in 15–30% of patients following acute cerebrovascular events, while approximately 40% are left with moderate functional impairments. The incidence and mortality associated with vascular pathologies continue to rise. These trends underscore the evolving strategies in the management of stroke patients, emphasizing the integration of advanced clinical, neurological, neurophysiological, and paraclinical diagnostic methods, refinement of differential diagnostics, optimization of somatic health, timely implementation of preventive measures, and overall reduction in disability and mortality resulting from acute cerebrovascular and cardiovascular conditions." Uzbekistan is no exception and occupies a leading position in this indicator. Every year, about 100-110 thousand people in our country suffer a stroke for the first time. The incidence of the disease is 282.3 thousand cases per 1,000 population, which significantly exceeds the average incidence rate for European Union countries - 200 cases per 100 thousand population, accompanied by a 2-fold increase in mortality.In foreign publications insultdan next psixoemosional disorders in the majority of patients (30-67%) observed in quotes if the patient 18-43% ida panic attacks have been recorded. “Insultdan next tserebrovaskulyar of a panic attack complications and associated with an increase in the risk of death is increased three times the risk of development of complications and death within 10 years tserebrovaskulyar increases several times".

Foreign publications indicate that psychoemotional disorders after stroke are observed in the majority of patients (30-67%), and panic attacks are noted in 18-43% of patients. “Panic attacks after stroke are associated with an increased risk of cerebrovascular complications and death, with mortality increasing threefold over

10 years, and the risk of developing cerebrovascular complications increasing several times."

A wide range of measures have been taken to develop the healthcare system of our country, including reducing the incidence of neurological diseases and their complications, as well as providing qualified medical care to patients with this disease, and tasks such as "...increasing the efficiency, quality and accessibility of medical care provided to the population, as well as forming a system of medical standardization, introducing high-tech methods of diagnosis and treatment, supporting and preventing a healthy lifestyle through the effectiveness of patronage services and dispensary visits..." have been set. These tasks determine priority areas such as raising the level of modern medical services in the diagnosis and treatment of complications of somatyc diseases among the population to a new level, and conducting in-depth scientific research on the use of modern technologies in the provision of quality medical services.

# I CHAPTER. REVIEW OF THE LITERATURE

## 1.1. Cognitive impairment and risk factors in patients with ischemic stroke.

Cerebral strokes are one of the global problems today due to their prevalence, increasing trend and serious complications, which have a significant impact on both human health and the country's economy. In economically developed countries, acute cerebrovascular accident (ACS) is the second leading cause of death after ischemic heart disease. ACS is the leading cause of disability. The mortality rate from cerebrovascular diseases is 5 times higher in the Russian Federation than in European countries, and the average age of stroke is 63.1 years in men and 66.3 years in women, which is ahead of Western countries (72.9 in men, 77.7 in women) (Feigin et al., 1995).

According to US scientists, a stroke occurs every 45 minutes, 700 thousand cases are registered per year, and patients die every 3 minutes, and the remaining 4.7 million survivors become disabled. Rehabilitation of the disabled requires huge economic costs (more than 50 billion US dollars annually). Approximately 60 thousand people in Uzbekistan suffer a stroke every year, and the level of disability is more than 80% (B.G. Gofurov 2006, Y.N. Madzhidova 2019).

The introduction of intensive care units into practice in the treatment of patients who have undergone acute circulatory disorders of the main mi, modern technological provision ensures the diagnosis of stroke and a decrease in mortality. Modern views on the treatment of this disease-pathogenetic treatment, stationary, rehabilitation centers affecting the mechanisms of the ischemic cascade, as well as

the correct conduct of treatment stages in outpatient conditions improve the indicator of the quality of life of patients.

"Stroke profoundly affects vital neurological functions, often leading to long-term health consequences. Statistically, more than half of individuals who suffer a stroke develop severe impairments, while around 30% remain dependent on others for basic care.

Globally, stroke is recognized as one of the most prevalent causes of disability, with an estimated 3.2 cases per 1,000 population. The resulting disability is primarily linked to post-stroke complications, which include motor impairments (in approximately 60% of cases), sensory disturbances, as well as disorders of speech, cognition (such as memory impairment and reduced cognitive efficiency), and emotional-volitional regulation."

In rehabilitation centers during the recovery period after acute cerebral circulation disorders, these patients are taught the necessary methods to adapt to external muxit, restore lost functions-movement and sensory disorders, restore speech disorders, but insufficient efforts to eliminate cognitive disorders even from common complications negatively affect the deterioration of the quality of patients post-stroke life and a positive course of the rehabilitation period.

Cognitive dysfunctions, like all pathological processes, are severe over time, and diseases that are not subject to correction, can turn into dementia. Five to fifteen percent post-stroke moderate cognitive impairment patients experience severe cognitive impairment after one year, and dementia after five years is observed in one hundred percent patients with post-stroke cognitive impairment. In conclusion, it is possible to say that all patients who have had a stroke belong to the risk group in which cognitive disorders develop.

"Arterial hypertension plays a significant role in the development of lacunar infarcts, particularly in deep brain structures such as the thalamus and basal ganglia, often resulting in diffuse alterations of the white matter. As reported by Y.A. Starchinoy and co-authors, cerebrovascular pathology is identified in approximately

73.7% of individuals diagnosed with hypertension. The coexistence of hypertension and stroke typically leads to more pronounced and earlier cognitive decline, predominantly manifested by impairments in executive function.

Although antihypertensive treatment can decrease stroke risk by nearly 45–50%, an excessive reduction in blood pressure may negatively affect cerebral perfusion, especially in the frontal cortical areas, thereby exacerbating cognitive deficits. Prolonged hypotension, particularly against the backdrop of advanced brachiocephalic artery stenosis, may provoke watershed-type ischemia and further aggravate neurocognitive dysfunction. In addition, atherosclerosis diminishes cerebrovascular reserve capacity, restricting the brain's compensatory response to changes in blood flow."

Data from the MONICA study (Multinational Monitoring of Trends and Determinants in Cardiovascular Disease) suggest that the presence of atherosclerosis alone does not fully account for cardiovascular complications, as it is observed in 15% of women and 40% of men without overt symptoms.

Emerging Risk Factors and Cardiac Comorbidities

In addition to traditional factors, elevated homocysteine levels (hyperhomocysteinemia) have emerged as a significant and independent predictor of early and progressive atherosclerosis. More than 80 clinical and epidemiological studies have confirmed its association with increased risk of thrombosis and vascular complications in coronary, cerebral, and peripheral arteries. These findings have led to the development of the homocysteine theory of atherosclerosis and its inclusion in classifications of thrombophilia.

Cerebral damage may also arise from cardiac pathologies, particularly those involving reduced cardiac output and hypoperfusion. Cognitive impairment is observed in 50–60% of elderly patients with chronic heart failure post-stroke cognitive impairments are a criterion for predicting an unfavorable functional outcome of ischemic stroke and have a negative impact on the daily activities and quality of life of patients;. Atrial fibrillation (AF), in particular, is highly prevalent

among older adults and is associated with increased morbidity, mortality, and cerebral microembolism. AF is considered an independent risk factor for post-stroke dementia and cognitive impairment due to its contribution to silent infarctions and cerebral hypoxia.

Thus, patients with comorbid hypertension, AF, and dyslipidemia are at substantially higher risk for developing post-stroke dementia. Recognizing these interrelated mechanisms is essential for developing effective prevention and treatment strategies.

**Clinical presentation and diagnosis of cognitive disorders in patients with ischemic stroke.**

Cognitive impairment refers to both subjective and objective declines in higher mental functions compared to the individual's baseline, resulting from organic brain damage. This decline affects performance in reading, work, daily tasks, and social engagement (Luria A. R., 1969; Yakhno N. N., 2003).

"Cognitive impairment following a stroke (PSCI) is often a direct consequence of the pathological processes initiated by the cerebrovascular event. Although such impairments may become apparent within the first month post-stroke, they most commonly develop over the course of the initial year. The primary etiological factor behind stroke is vascular pathology, involving structural and functional damage to large or small-caliber vessels, which disrupts cerebral microcirculation. The scale of ischemic injury is largely determined by the extent of vascular compromise, with major arterial lesions typically resulting in extensive zones of cerebral infarction."

The type and intensity of cognitive decline are directly influenced by the affected brain area and the size of the ischemic zone. For example, damage to large-caliber arteries often results in a broader range of cognitive deficits, while involvement of smaller vessels tends to produce more uniform neuropsychological symptoms. Researchers emphasize that the severity of cognitive dysfunction is

strongly associated with the level of structural brain damage—the deeper or more critical the area, the more pronounced the cognitive decline.

"Mild cognitive impairments are more frequently observed when integrative brain systems are affected. Beyond the size and anatomical localization of the lesion, a range of additional factors significantly influence recovery potential. These include the patient's age, the functional capacity of cerebral compensatory mechanisms—such as brain reserve and neuroplasticity—as well as the condition of the vascular system and the degree of neuronal maturity."

According to A.R. Luria (1962), higher mental functions rely on intricate systems involving multiple, sometimes distant, interconnected brain regions. These functions are not confined to narrow areas of the cortex or isolated neuron groups. Supporting this view, researchers such as Filimonov I. N. (1940, 1974) and Pavlov I. P. (1951) emphasized the functional flexibility of brain regions. They proposed that under certain conditions, specific brain areas can assume new roles, a concept foundational to the theory of neuroplasticity—the brain’s ability to reorganize and adapt the localization of cognitive functions.

Stroke, like other forms of brain injury, can activate mechanisms of neuroplasticity and neural repair. These processes are vital for compensatory recovery, enabling functional reorganization of preserved areas surrounding the damaged tissue.

Post-stroke cognitive impairment generally falls into three main clinical and pathophysiological categories:

1. Acute-onset cognitive impairment with a regressive or stable course – typically follows a single stroke affecting a "strategic" brain region responsible for cognition. This type often presents with a monophasic clinical trajectory.

2. Focal brain damage affecting isolated functional regions – seen in a small percentage of cases (under 5%). These rare cognitive disorders vary according to lesion location. For example, injury to the optic chiasm may result in severe memory deficits (similar to Korsakoff’s syndrome), reduced initiative, and hallucinations.

Lesions in the head of the caudate nucleus may lead to cortical-subcortical disconnection, impairing frontal lobe activity and visual memory processes.

3. Cognitive decline due to decompensation of pre-existing cerebrovascular disease – this is the most common clinical pattern. In such cases, stroke exacerbates already compromised brain structures, worsening cognitive performance over time.

This variant is characterized mainly by impaired "executive" functions, memory impairment is also noted. According to some authors, the above disorders are observed in 40% of patients with stroke.

"Executive dysfunction is considered the predominant form of cognitive impairment not only in the post-stroke period but also in various other cerebrovascular pathologies. This is primarily associated with the disconnection between the frontal lobes and other brain regions, which arises from diffuse white matter damage, known as leukoaraiosis.

Consequently, cognitive decline in such cases is driven not only by acute focal lesions, such as those resulting from stroke, but also by chronic progressive cerebrovascular insufficiency."

2. "Cognitive impairments resulting from mixed (vascular-degenerative) brain damage are observed in 10-30% of post-stroke cognitive disorders. Diagnosing mixed forms of cognitive dysfunction presents specific challenges, particularly in patients with established cerebrovascular pathology where post-stroke cognitive decline is often detectable. Notably, the progressive nature of cognitive deterioration plays a crucial role in diagnosis.

Research indicates that many 'pure' post-stroke cognitive impairments tend to improve within the first few months, with only 10% of patients experiencing persistent cognitive decline over time. Cases of purely vascular cognitive impairment following stroke are rare, as neuroimaging studies often reveal 'silent' infarctions, leukoaraiosis, and atrophic brain changes at the onset of cognitive deficits, suggesting a combination of vascular damage and neurodegenerative processes."

It is believed that temporal relationships are not always associated with causal relationships (for example, memory impairment in patients after stroke is not as pronounced as in Alzheimer's disease). In such cases, it is necessary to take into account the history of the disease, the existing risk factors for vascular pathology, the nature of the disease and its course, and neuroimaging data. Damage to any brain region can lead to similar cognitive impairment or behavioral disorders (Henry J. Riordan, 2011). For example, many authors have noted that ischemic and hemorrhagic strokes of the tegmental area and adjacent medial temporal lobe structures can cause a wide range of cognitive impairments, but the most common impairments include auditory, visual perception, and memory impairment.

### 1.3 Post-stroke cognitive impairment with panic attacks

Panic attacks are a nosological category that includes panic attacks that develop with a certain frequency, the diagnostic criteria for which are established. A panic attack is a unique set of symptoms characterized by paroxysmal anxiety that cannot be confused with other psychological phenomena. The diagnosis of a panic attack does not cause difficulties. According to the ICD-10 criteria, a panic attack is characterized by paroxysmal fear (often accompanied by a feeling of imminent death) or a feeling of internal tension in combination with four or more of the symptoms listed in the list of symptoms associated with panic As a result of improving the diagnosis and treatment of post-stroke panic attacks based on the obtained scientific results on the prevention of panic attacks in patients with stroke, the following methods were developed and implemented:

Methodological recommendations "Assessment and prognosis of cognitive changes accompanied by panic attacks in patients who have suffered a stroke "(Certificate No. 04-r / 9 dated 23.02.2023 of the Tashkent State Dental Institute).

The implementation of the proposed methods will increase the economic, medical and social effectiveness of treatment, reduce the number of patients at risk 46 of developing panic attacks in ischemic strokes, as well as the number of patients

who bear high costs as a result of the disease, which, in turn, will lead to an improvement in the quality of life of this category of patients;

Scientific results of methods for optimizing the diagnosis and treatment of patients with stroke are included in the practice of healthcare, including the clinical practice of the multidisciplinary clinic of the Tashkent Medical Academy and the neurological Department of the Clinical Hospital no. /19-issue 06.09.2023) As a result of the implementation of the proposed methods, positive results were achieved in terms of correct diagnosis of patients with panic attacks, optimization of therapy, prevention cognitive impairment associated with panic attacks after ischemic stroke, improving the quality of life of patients.. . The list of symptoms associated with panic includes the following: its peak within 10 minutes, unlike most paroxysmal states, a panic attack is not characterized by a prodromal period (aura). The period after the attacks is characterized by general weakness, fragility. The duration of a panic attack is several minutes, with an average of 15-30 minutes. Although some patients report longer attacks. The frequency of attacks varies greatly in the patient population. Attacks can occur as isolated events, and at the same time their frequency in individual patients reaches several attacks per day.

The name of paniticks attacks is now recognized worldwide thanks to the classification of the American Neuro-psychiatric Association. In 1980, it is based on the fact that complex treatment of cognitive disorders in patients with ischemic stroke improves the quality of life of patients with post-stroke cognitive disorders, reduces the severity of neurodynamic, regulatory and operational cognitive disorders, and reduces the severity of affective disorders. , members of this association proposed a new manual for diagnosing mental disorders, the DSM-III-R, which was based on clear, mainly phenomenological criteria. In the latest version of this manual, the diagnostic criteria for VH are as follows

- as a result of a comprehensive assessment of neuropsychological
- functions in patients with ischemic stroke,
-in addition to the heterogeneity of the profile of cognitive disorders

-after stroke,

-the relationship between cognitive disorders,

- daily activities and the quality of life of

-patients was scientifically substantiated;

It has been proven that a high rate of cognitive impairment in patients with ischemic stroke is associated with subclinical brain damage detected by CT and MRI in the form of diffuse white matter lesions, heart attacks in silent areas of the brain, and brain atrophy;

post-stroke cognitive impairments are a criterion for predicting an unfavorable functional outcome of ischemic stroke and have a negative impact on the daily activities and quality of life of patients;

It is based on the fact that complex treatment of cognitive disorders in patients with ischemic stroke improves the quality of life of patients with post-stroke cognitive disorders, reduces the severity of neurodynamic, regulatory and operational cognitive disorders, and reduces the severity of affective disorders.

The occurrence of panic attacks is not due to the direct physiological effects of any substances (for example, drug abuse or medication, or somatic diseases, such as thyrotoxicosis).

Thus, panic attacks consisting of four or more symptoms [99, 102, 105] are considered to be autonomic paroxysms, seizures. Panic attacks (PA) are the most specific category of panic disorders among neurological diseases. The prevalence of panic attacks in the general population is ~1.9% -3.6%; the addition of agoraphobia and social adaptation leads to the loss of jobs of patients, which allows us to consider this pathology not only as a medical, but also as an important socio-economic problem.

"In neurology, the study of panic attacks (PA) has traditionally focused on understanding the interplay between biological and psychogenic factors in their development. Several theories explore the mechanisms underlying panic disorder, including cognitive-behavioral models, which form the basis for treating panic

attacks. A concise integration of these theories views panic attacks as a series of escalating anxiety responses, disrupted information processing (characterized by catastrophic thinking), and the adoption of maladaptive coping strategies (such as safety behaviors and escape rituals) aimed at alleviating fear, reducing anxiety, and restoring a sense of security. Individuals experiencing panic attacks tend to have heightened anxiety levels, frequently fixating on their physical sensations, struggling to assess them rationally, and failing to recognize them as non-threatening." (22).

"Anxiety and depressive disorders, including panic attacks, are more prevalent in women than in men. The higher incidence of anxiety disorders among women is attributed to both biological and sociocultural factors, particularly the cultural acceptability of anxiety in women. Despite consistent epidemiological evidence indicating a higher prevalence of anxiety disorders in the female population, with a ratio ranging from 1.4 to 5.4, the impact of key biological factors such as gender, along with psychosocial influences like role behaviors, on the development of panic attacks remains insufficiently explored. The term 'gender' refers to personal characteristics associated with masculinity and femininity, largely shaped by sociocultural influences, known as psycho-behavioral patterns. Differences between men and women in certain emotional responses are largely determined by varying levels of masculinity and femininity. Contemporary shifts in gender roles have highlighted that many gender differences are not biological in nature but stem from cultural and socialization processes. Gender influences various aspects of life, including the evaluation of personal resources, coping mechanisms in response to stress, societal interactions, self-esteem, and expectations of others. Strong evidence suggests that gender role behaviors may be linked to the development and persistence of anxiety and fear symptoms. Exploring subtle behavioral patterns that can positively or negatively affect the health of individuals with panic attacks is an important and under-researched area that warrants further attention."

### 1.4 Cognitive impairment prevention and treatment strategies.

"Age is recognized as a primary and independent risk factor for the onset of cognitive impairments. Involutional changes in brain structure, including reductions in brain volume and neuronal loss associated with aging, play a significant role in the development of cognitive dysfunction. Middle-aged individuals generally exhibit a lower risk of progressing to dementia, and their cognitive deficits tend to be temporary. This is explained by the concept of 'cognitive reserve,' which refers to the brain's ability to compensate for damage through neurons that can form new synaptic connections to maintain cognitive function. Consequently, in a variety of cerebrovascular or neurodegenerative disorders, clinical symptoms may remain undetected for prolonged periods. However, once the brain's compensatory mechanisms are exhausted, cognitive deficits become more pronounced, particularly when extensive lesions are present."

"Degenerative changes in the brain tissue also influence the severity of cognitive deficits observed following a stroke. According to some researchers, such impairments may represent a prodromal phase of dementia, as approximately 32% of post-stroke patients with cognitive dysfunction exhibit disturbances in memory trace consolidation. In these instances, a mixed etiology involving both vascular and neurodegenerative brain damage cannot be ruled out."

Atherosclerosis, diabetes mellitus, arterial hypertension, and both macro- and microangiopathies are recognized to induce morphological alterations in the brain's white matter—such as demyelination and gliosis—which, over time, disrupt communication between cortical and subcortical brain structures and contribute to cognitive decline. Diabetes mellitus, in particular, is considered an etiological factor in post-stroke dementia, as it has been shown to double the risk of its development. Research indicates that 71% of elderly patients with type 2 diabetes and panic attacks exhibit cognitive impairments, irrespective of concomitant cardiovascular diseases. Among these individuals, 51% presented with moderate cognitive dysfunction, while 20% experienced mild cognitive deficits [104].

"Examination findings indicate that patients who have experienced a first stroke and score well on the MMSE should implement preventive measures to

mitigate the risk of subsequent cognitive deterioration and recurrent strokes. Additionally, continuous cognitive monitoring and neuroprotective treatment are advised, especially for those with lower MMSE scores. [143, 145, 147, 148, 175].

In addition to motor, speech, and cognitive impairments, stroke patients frequently experience a range of psychoemotional disturbances, including depression, anxiety, asthenia, apathy, impulsivity, negativism, diminished euphoria, and reduced emotional stability (Kim J.S., 2000; Kadykov A.S. et al., 2009). Among the various post-stroke complications, psychoemotional disorders—particularly anxiety and depressive states—are the most prevalent. These conditions significantly hinder rehabilitation outcomes and delay patients' reintegration into their normal social environments (Gaete J.M., Bogousslavsky J., 2008). Moreover, the presence of psychoemotional disorders negatively impacts both patients and their caregivers, reducing treatment compliance and overall quality of life." (Kim J.S., 2016).

The comorbidity of stroke and anxiety-depressive states mutually burdens the clinical manifestations of each case and contributes to the development of the developed pathological process. Laboratory and co-authors M.L.C.Labi (1980) considered VH as a rarely recognized complication of stroke, and it remains so today (Kim J.S., 2016), often VH is not noticed by neurologists.

"This review explores the phenomenology of common psycho-emotional disorders, including mood and emotional disturbances, that occur following a stroke, as well as the pharmacotherapeutic options available, particularly the use of selective serotonin reuptake inhibitors (SSRIs). Psycho-emotional disorders are observed in the majority of stroke patients, affecting 30-67% of individuals. Among these, anxiety-depressive states are noted in 18-43% of patients (Verdelho A. et al., 2004). Post-stroke depression is generally associated with an elevated risk of cerebrovascular complications and increased mortality. Specifically, the mortality rate triples over a 10-year period (Townend B.S. et al., 2007), and the risk of cerebrovascular events is significantly heightened: myocardial infarction by 4.5%,

stroke by 2.7%, and complications related to type 2 diabetes mellitus by 2.2% (Skvortsova V.I. et al.)."2009).

In addition to the obvious threat of complications leading to death in the case of depression, other psychoemotional disorders, especially when it comes to the social adaptation of patients (Gillen R. et al., 2011), decrease the quality of life (Liman T.G. et al., 2012). In addition, untimely recognized psychoemotional disorders significantly reduce the effectiveness of rehabilitation efforts (Gillen R. et al., 2011). The location and volume of brain damage in stroke is also a significant factor. In this regard, the topic of the relationship between the localization and volume of the stroke focus and their influence on the development of post-stroke depression is very relevant. The "left frontal lobe" theory was proposed (Robinson R. G. et al., 1984), which explains the high frequency of severe depression in the acute period of stroke with localization of the focus in the left hemisphere, involving the dorsolateral cortical area and the left basal nuclei. In this regard, the involvement of the fronto-basal ganglion pathways of the brain in post-stroke depression and changes in neurotransmitter systems in the anterior frontal lobe damage play a special role (serotonergic, adrenergic and dopaminergic systems). An important discovery in recent years has been the suppression of neurogenesis under the influence of hypercortisolemia with a decrease in the level of brain-derived neurotrophic factor (BDNF) and long-term depression, leading to the development of brain atrophy and cognitive dysfunction, and proving the importance of antidepressant treatment (Martinowich K., Lu B., 2007). Thus, the general strategy for treating patients with post-stroke psychoemotional disorders is based on the role of drugs that modulate neurotransmitter systems and increase BDNF levels, in particular antidepressants (Jorge R.E. et al., 2010).

"Unfortunately, to date, there are no large-scale controlled trials demonstrating the effectiveness of specific treatment methods in preventing or mitigating post-stroke cognitive impairment. However, it is universally acknowledged that preventing further damage to the brain tissue and recurrent strokes is crucial. Several measures are employed to achieve this, primarily focusing on the effective

management of cardiovascular and cerebrovascular risk factors, such as arterial hypertension (through antihypertensive agents), hyperlipidemia (via diet and statins), and hyperhomocysteinemia (using vitamins B6, B12, and folic acid) [8, 231].

Numerous studies have indicated that appropriate management of arterial hypertension in stroke patients not only prevents recurrent strokes but also reduces the risk of developing dementia." [168].

Antiagregant drugs or anticoagulant drugs can be used to prevent the occurrence of recurrent ischemic stroke (high risk of cardiogenic embolism or coagulopathy). However, patients with neurovisiulation symptoms of cranial microangiopathy, especially extensive subcortical leukoareosis and microgemorrhagia (detected in a special MRI mode–gradient Exo T2*-in weighted images), should be noted for the association with the administration of anticoagulant drugs and high doses of antiagregant drugs. It should also be taken into account that should not be used when there is a high risk of intraserebral bleeding [38].

To improve cognitive activity in patients, a different assortment of nootropic drugs is used, which can be divided into 4 main groups: 1) drugs that affect the neurotransmitter systems, 2) drugs that affect the trophic level of nerve tissue, 3) drugs with neurometabolic effects 4) drugs with vasoactive effects.An important problem is that for most drugs used in domestic clinical practice, there are no data from placebo-controlled studies that reliably confirm their effectiveness.

"Controlled studies have demonstrated that a clinically significant placebo effect can occur in 30-50% of patients with cognitive impairments, even in those with severe dementia. Furthermore, considering the tendency for cognitive function to spontaneously improve during the early recovery phase post-stroke, it becomes more challenging to substantiate the positive effects of pharmacological interventions following a stroke."

Controlled studies in patients with vascular dementia have shown the effectiveness of drugs belonging to the first group and mainly affecting the

cholinergic system (cholinesterase inhibitors, for example, galantamine or rivastigmine), as well as the glutamatergic system (NMDA-glutamate receptors memantine) - a type of stroke in vascular dementia, the effectiveness of which is not specifically assessed in controlled studies. On the other hand, cholinesterase inhibitors and memantine have shown significant effects in some patients with vascular dementia, but their efficacy is considered moderate [25, 31].

Cerebral stroke is an acute emergency that impairs the brain's compensatory ability. Ischemic stroke, the most common form, can be permanent or progressive. After stabilization, it is crucial to monitor for potential episodes of blood supply decompensation. These episodes can persist for years and lead to irreversible changes in brain tissue, diminishing its functional activity [55, 71, 113, 187]. The ischemic cascade underlying this process has been extensively studied, and an algorithm for treating ischemic stroke (IS) patients has been developed. Research shows that the acute phase of a stroke consists of two successive stages: neuropathy (lasting up to 6 hours) and neurorepair (beginning after 6 hours). The neurorepair phase can last for months or even years, significantly influencing drug therapy choices in patients with neurological and psychological disorders (NMSD) [6, 64, 83, 84, 92, 115-117].

Thrombolytic therapy using fibrinolytic agents, such as recombinant tissue plasminogen activator, has introduced new possibilities in the treatment of thrombotic stroke. However, this method has limitations and is not yet widely available [6, 71, 72, 150, 182]. Furthermore, studies have not demonstrated a significant positive impact on cognitive function or functional outcomes six months post-stroke [150].

"The primary goal in managing patients with acute cerebrovascular accidents is neuroprotective therapy, which aims to safeguard neurons affected by hypoxia, thereby preserving their function—particularly crucial during the acute phase of a vascular event when the brain’s compensatory mechanisms are compromised, and its reserves are exhausted. In addition, neuroprotective agents facilitate the repair processes in nervous tissue [47, 61, 69, 72, 84, 115, 116]. Although this modern

therapeutic approach has led to positive outcomes, such as enhanced survival rates and improved quality of life, certain limitations in the research findings still necessitate further exploration and refinement."

Currently, there is a wide range of pharmacological agents recognized as effective in the treatment of patients with NMSC. Researchers suggest various treatment protocols, taking into account the pathogenesis of the condition and the stage of the processes during the acute phase of stroke.

The discovery of neuroplasticity, which accompanies any brain injury, has significantly altered the approach to treating patients with NMSC. Experimental evidence has confirmed the role of neuroplasticity in the structural and functional recovery of neurons, as well as the activation of compensatory and restorative mechanisms in surviving neurons. Since most neurological diseases progress through distinct stages, neuroplastic mechanisms can compensate for the manifestation of focal neurological symptoms in the early stages, thereby alleviating their severity. [49, 85, 180, 182].

It is known that brain stroke itself is a catalyst for neuroplastic processes. Nervous tissue is able to rebuild under the influence of harmful factors to restore brain activity when connections between neurons are broken (synaptic plasticity). However, there are limitations in the process of brain tissue reorganization: the patient's age, the severity of cerebrovascular insufficiency before the stroke, etc. [55, 64, 157, 158, 170, 186, 187].

The brain's compensatory abilities and the reversibility of functional and morphological changes are influenced not only by the presence of risk factors and comorbid conditions but also by factors such as the extent of the lesion, the patient's age group, and the initial condition of the brain tissue [46, 47, 134, 135, 147, 161]. The use of neuroprotective therapy with peptide-based drugs has been extensively studied. These therapies offer broad protection to the brain from harmful factors, support the restoration of neurological functions by reorganizing neuronal connections, and reduce the brain's energy expenditure during hypoxia. It has been

established that organ-specific active compounds, such as neuropeptides found in organ extracts (e.g., heart, brain, and liver extracts from animals), are present in such preparations and are most effective at low concentrations in the context of a given pathological process [120, 121]. During the acute phase of the pathological process, neurotrophic factors that are essential for neurons regulate the proliferation, survival, migration, and differentiation of nervous system cells, ensuring their survival and proper functioning. [49, 70, 120, 121].

In addition, neuroprotective therapy usually has a general application, which allows it to be used in various subtypes of NMSD in the prehospital stage, and its timely appointment improves the rehabilitation potential of patients.

A local drug of the neuropeptide group (tricortin), which has undergone numerous clinical trials and has proven its efficacy and safety in many neurological diseases, is one of the few currently available drugs, which allows it to be used not only in the treatment of patients with ischemic and hemorrhagic stroke, but also in emergency neurology with brain edema [68, 93, 95]. The experience of using Tricortin in the treatment of ischemic and hemorrhagic stroke is about 10 years [11,34, 65, 92, 94, 111].

A multicenter, double-blind, randomized, placebo-controlled study (escort study) is of particular note: the use of the drug at a dose of 1000 mg per day in the acute and early recovery period of ischemic stroke (IS); neurological regression was observed in the acute period, compared with placebo, in patients with moderate and severe neurological deficits, focal symptoms and improvement of cognitive functions from the first days of therapy; the early recovery period is characterized by full independence in daily activities of patients and 100% recovery of cognitive functions in patients with low initial scores on the MMSE scale.

"The high efficacy and safety of a low-dose local neuroprotective drug, administered through a single or repeated ten-day course, were demonstrated in patients with ischemic stroke when compared to a placebo. Two treatment courses with the local neuropeptide drug led to sustained positive changes in neurological

status (NIHSS), recovery of daily functional activities (Rankin, Barthel, Rivermead scales), and improvements in cognitive functions (MMSE). As a result, patients exhibited strong rehabilitation potential during the early recovery phase following ischemic stroke." (Stakhovskaya L. V. et al. 2012) [108].

## CHAPTER II. PERSONAL EXAMINATION RESULTS

### 2.1. Clinical characteristics of patients

"During the acute phase, mild hemiparesis was observed in 83 (73%) patients, characterized by a reduction in muscle strength by up to 4 points. Moderate hemiparesis was found in 31 (27%) patients, with muscle strength reduced by up to 3 points in at least one limb. Movement disorders were more pronounced in the arm, particularly in the proximal portion. Additionally, central paresis of the facial muscles on the affected side and deviation of the tongue tip towards the side of paresis were noted."

Sensitivity disorders in the form of hemihypesthesia were detected in 92 (81%) patients.

Speech disorders were detected in 50 (48%) patients, of which 36 (32%) patients had dysarthria and 14 (12%) patients had mild motor aphasia.

General cerebral symptoms were poorly expressed. All patients were conscious during the examination. 31 patients (27%) complained of moderate temporary headache, of which 9 (7%) had diffuse headache, 11 (9%) had it localized to the side of the lesion, and 10 (9%) had nausea and dizziness.

Thus, patients with relatively mild neurological deficits were included in the study.

The NIHSS scale index (Appendix 2) correlated with systolic blood pressure ($r=0.31$, $p<0.5$), diastolic BP ($r=0.28$, $p<0.5$), and mean BP ($r=0.33$, $p<0.5$) on the first day of stroke.

Patients with long-term arterial hypertension had higher blood pressure levels than patients without arterial hypertension ($p<0.05$) (Table 2.1).

**2.1.Table**

**Blood pressure levels on the first day of stroke in patients with and without pre-existing arterial hypertension**

| The electoral "lapses" the level of | arterial hypertension inpatients yali n=75 | Arterial hypertensionpatients yas |
|---|---|---|
| Sistolaelectoral "lapses" | 189,9±25,9 | 161,0±14,5* |
| Diastolikelectoral "lapses" | 103,1=1=11,0 | 96,0±7,0 |
| Pulseelectoral "lapses" li, | 87,0±20,0 | 65,0±11,8* |
| electoral "lapses" average | 132,2±14,6 | 117,7±8,5* |

* - statisticaldifferences reliable

In patients with coronary heart disease, blood pressure on the first day of stroke was higher than in patients with cardiovascular disease ($p<0.05$) (Table 2.2).

NIHSS scores did not differ significantly in patients with pre-existing arterial hypertension (4.4±1.1 points) and those without arterial hypertension (4.4±1.1 points); in patients with cardiovascular disease (4.5±1.1 points) and those without cardiovascular disease (4.1); with cardiovascular disease (4.1±0.9 points); with cardiovascular disease with signs of heart failure (4.4±1.1 points) and without signs of heart failure (4.3±1.1 points); with diabetes mellitus (4.4±1.2 points) and without diabetes mellitus (4.4±1.1 points).

**2.2.Table**

Blood pressure levels on the first day of stroke in patients with and without coronary heart disease

| The electoral "lapses" the level of | patients have found yu n=55 | patients who do not have yu n=20 |
|---|---|---|
| Sistolaelectoral "lapses" | 188,5±27,1 | 179,3±24,1* |
| Diastolikelectoral "lapses" | 102,9±11,5 | 100,0±8,6 |
| Pulseelectoral "lapses" | 85,6±19,8 | 79,8±22,3* |
| Pulseelectoral "lapses" | 131,0±15,7 | 126,6±11,7* |

* - reliable statistical differences

**2.2. CT/MRI data of patients participating in the study**

All patients had focal brain lesions consistent with the clinical presentation of ischemic stroke on CT or MRI, which was an inclusion criterion. Ischemic lesions were found in the right hemisphere in 50 (44%) patients, in the left hemisphere in

64 (56%). Subcortical localization of the lesion was observed in 67 (59%) patients, in the frontal lobe in 6 (0.5%), in the parietal lobe in 12 (11%), in the temporal lobe in 13 (12%), and in 16 (14%) patients, preexisting lesions were detected, of which 8 (50%) patients had small lesions, 6 (37.5%) patients had medium lesions, and 2 (12.5%) patients had large lesions.

The distribution of identified foci of cerebral infarction according to the vascular basin and focus localization is presented in Table 2.3.

**2.3 jadval**

Cerebral infarction location centre

| Havzal | the right half of the ball | the ball in the left half of | the total amount of |
|---|---|---|---|
| the brain artery in the previous | 2 | 4 | 6 |
| middle cerebral artery | 25 | 35 | 60 |
| Front xoro artery | 6 | 8 | 14 |
| total amount | 33 | 47 | 80 |

Diameter 3-14 mm , which is small foci of 23 persons (28%) patients, in diameter, 15-30 mm , which is secondary foci of 40 students (50%) patients, with diameter 30 mm than than which is large foci of 17 persons (22%) in patients was detected.

The brain is not even detected by the oven size and blood vessels into the pool of looking distribution 2.4-in the table are listed.

**2.4. Table**

The brain is not even detected by the oven size and blood vessels into the pool of looking distribution

| the pool is | Tiny section | average size section | Large section |
|---|---|---|---|
| Front brain artery | 3 (13%) | 2 (6%) | 1 (5%)of |
| the middle cerebral artery | in 15 (65%) | 23 (72%) | 17 (85%) |
| Front xoro artery | in 5 (22%) | 7 (22%) | 2 (10%) |
| the total amount | of 23 (31%) | 32 (43%) | 20 26%) |

Leukoaraiosis was detected in 46 (57.5%) patients as areas of decreased density on CT or increased signal intensity on T2-weighted brain MRI, with a mean visual rating scale score of 0.8±0.8. Of these, mild periventricular and/or punctate subcortical leukoaraiosis <10 mm in width was detected in 31 (67%) patients, moderate, patchy, partially confluent subcortical leukoaraiosis was detected in 13 (29%) patients, and subcortical leukoaraiosis >10 mm in width, with a combined width of >20 mm in 2 (4%) patients.

Outer cerebral atrophy in the form of widening of cortical sulci was detected in 64 (80%) patients, with a mean visual rating scale score of 1.1±0.7. Of these, 50 (78%) patients had mild atrophy, 12 (19%) had moderate atrophy, and 2 (3%) had severe atrophy.

Ventricular dilatation was detected in 73 (91%) patients, with a mean score on the visual rating scale of 1.5±0.6. Of these, mild was noted in 38 (52%) patients, moderate in 33 (45%) patients, and severe in 2 (3%) patients.

A correlation was found between the prevalence of leukoaraiosis and age ($r=0.37$, $p<0.05$). The severity of leukoaraiosis was associated with SBP ($r=0.26$, $p<0.05$), DBP ($r=0.27$, $p<0.05$), and mean blood pressure ($r=0.23$, $p<0.05$) in the acute period.

Leukoaraiosis was more common in patients without arterial hypertension

(0.9±0.8 points, $p<0.05$), in patients with heart failure (1.1±0.8 points, $p<0.05$), in patients without signs of heart failure (0.6±0.6 points, $p<0.05$), and in patients with cardiovascular disease (1.1±0.8 points, $p<0.05$) than in patients without cardiovascular disease (0.5±0.5 points, $p<0.05$).

There was no correlation between leukoaraiosis and NIHSS scores in the acute period.

Leukoaraiosis was associated with the degree of ventricular system dilatation ($r=0.43$, $p<0.05$), but not with the severity of outer cerebral atrophy.

The degree of ventricular system dilatation and the severity of outer cerebral atrophy were associated with age ($r=0.25$, $r=0.33$, $p<0.05$, respectively).

"Ultrasound examination of the carotid arteries identified vertebral artery pathology in 26 (33%) patients. However, these findings were not correlated with clinical, neuropsychological, or neuroimaging data. The ultrasound duplex scanning of the carotid arteries revealed no stenosis in 35 (44%) patients. Mild stenosis of the external or internal carotid arteries, with a vessel diameter narrowing of up to 30%, was observed in 20 (25%) patients. Moderate stenosis of the internal carotid arteries, with narrowing ranging from 30% to 70%, was present in 15 (19%) patients, while severe stenosis, defined by a narrowing of more than 70%, was noted in the internal carotid arteries of other patients."

17 patients (43%) had internal carotid artery stenosis, 18 (45%) had right internal carotid artery stenosis, 3 (7%) had external carotid artery stenosis, and 2 (5%) had bilateral internal carotid artery stenosis. Of these, 31 (93%) patients had hemodynamically insignificant stenosis, and 3 (7%) had hemodynamically significant stenosis.

"The levels of blood pressure and NIHSS scores upon admission were not found to be correlated with the degree of stenosis. Stenosis was most frequently observed in patients with cardiovascular diseases. Among patients with coronary

artery disease, mild carotid artery stenosis was found in 12 (15%) patients, moderate stenosis in 8 (10%) patients, and severe stenosis in 7 (8%) patients.

In patients with arterial hypertension, mild stenosis was detected in 8 (12%) patients, moderate stenosis in 4 (6%) patients, and severe stenosis in 1 (2%) patient."

### 2.2 Analysis of cognitive disorders in examined patients.

One month after the stroke, according to the results of neuropsychological studies, cognitive impairments of varying severity compared to age standards were detected in 58 (73%) patients. The average assessment of cognitive functions according to the Cockman scale in the entire group was 33.2±4.3 points 1 month after the stroke. 51 (64%) patients complained of memory loss, apathy, decreased mental performance, incoordination, impaired spatial orientation, rapid fatigue, and difficulties in arithmetic operations. 39 (76%) of them had cognitive impairments of varying degrees, 12 (24%) patients had cognitive functions within the age norm. At the same time, 19 (33%) patients with cognitive impairments and 5 (29%) patients with cognitive functions within the age norm did not complain of such complaints.

In 17 (23%) patients, cognitive functions were within the age norm or reduced by several standard deviations from the age norm, their Cockman scale scores ranged from 36 to 38 points and averaged 37.5±1.5.

Moderate cognitive impairment was diagnosed in 47 (59%) patients. Their Cockman scale scores ranged from 29 to 37 points and averaged 34.0±3.1 points.

Definite cognitive impairment reaching the level of dementia was detected in 11 (14%) patients after 1 month. Cockman scores in these patients ranged from 22 to 28 points and averaged 25.6±3.5 points (Table 2.5).

Cognitive functions were assessed using the MMSE (Appendix 1) and MoCA-test method (Appendix 2), the Khachinsky scale (Appendix 3), and the Namilton scale for assessing panic attacks developing after a stroke.

Qualitative analysis of neuropsychological data in patients showed that they

were mainly characterized by mild or moderate neurodynamic disorders, manifested mainly by slowness, spontaneity, decreased performance, rapid fatigue, difficulty in starting a task, and impaired coordination functions.

Deviations during the "coding" test were detected in 65 (81) patients. Decreased indicators were detected in the "repeating numbers in forward order" test in 15 (19%) patients, and in the "repeating numbers in reverse order" test in 48 (60%) patients.

Visual memory test impairment was detected in 68 (85%) patients. Of these, 33 (49%) patients showed deviations from the age norm with direct retrieval, 58 (85%) patients with delayed retrieval.

Auditory-speech memory impairment was detected in 67 (58%) patients on the "5-word recall" test.

Hippocampal type of memory impairment (with impaired medial retrieval and recognition) was detected in 19 (24%) patients, and frontal type (with impaired retrieval, but with more preserved recognition and medial retrieval) in 52 (65%) patients.

**2.5. Table**

Proportion of patients with various cognitive impairments 1 month after stroke

| Cognitive disorders in | the total number of patients, % n=80 | Average cognitive impairment, % n=69 | Dementia, % n =11 |
|---|---|---|---|
| Carefully disorder | 81,1 | 57,3 | 100 |
| Memory disorder viziual, hearing | | | |
| - visual | 72,3 | 52,1 | 91 |
| - eseth | 62,4 | 43,1 | 100 |
| visual-spatial disorders | 42,1 | 31,4 | 73 |
| speech disorder | 80,2 | 69,2 | 100 |
| semantic associations | 68,1 | 31 | 91 |

| | | | |
|---|---|---|---|
| fonetikk association | 80,6 | 62 | 100 |
| grammatical associations | of 51.1 | 40 | 91 |

"The graph of the hour" in the test due to the deviation of 34 students (45,3%) in patients detected, this test on 3 points from 9 points, the average score 7,7±1.5 points. "The graph of the hour" of the test a violation of the task a holistic picture to create and store the possible lack, as well as the actions of the sequence of planning and implementation meetings holding defects with associated eDi.

64 students (85,3%) patients in the emay be rk and directed associations for test speech active mild or moderate decrease recorded ebuilt in the us. Of them, 55 students (73,3%) in patients with semantic mediation assosiasiya of the number reduced, 64 students (85,3%) in patients fonetik speech activity for the test in deviation, 41 students (54,7%) in patients grammatical intermediary assosiasiya of the number reducedwill determine of gan.

Average cognitive defects of the patients of 85 percent attention disorder, patients of 66.7 percent seen in the memory of disorder, 77,3 percent eseth to-speech, memory disorder, of the patients 47 percent seen-spatial disorders and patients with 91 percent of speech activity is impaired. Dementsiya with who are suffering from conditions of patients with 100 percent attention of a violation, eseth to-speech , memory and speech activity is detected, patients, 91 percent visual memory disorder, patients with 73 percent visual-spatial disorders identified.

Strokefrom 1 month later, various different cognitive disorderswork available patients , the number of 2.5-in the table are listed.

**2.6- Table**

Distribution of patients according to the degree of cognitive impairment according to the localization of the ischemic focus

| The patient group | of the oven right coronary-localization (%) | Left-sided localization of the ischemic focus (%) |
|---|---|---|

| | | |
|---|---|---|
| cognitive disorders | 8 (24,2%) | 10 (23,8%) |
| Average cognitive breaksshwith s | 19 (57,6%) | 26 (61,9%) |
| Dementia | 6 (18,1%) | 6 (14,3%) |
| total | 33 (100%) | 42 (100%) |

Thus, mainly neurodynamic and regulatory disorders were detected in most patients.

According to the results of questionnaires conducted among patients and their relatives, 19 (25%) patients had pre-stroke cognitive disorders in the form of memory loss, difficulties in visual-spatial orientation, complaints of weakness and reduced calculation. A hippocampal type of memory impairment was found in all patients with premorbid cognitive impairment.

Depending on the localization of the ischemic focus, the distribution of patients according to different levels of expression of cognitive impairment is presented in Table 2.6.

Thus, in patients with left-sided localization of the ischemic focus, compared to patients with right-sided localization of the ischemic focus, average cognitive impairment was detected, but this difference did not reach the level of statistical reliability.

"One month post-stroke, patients with ischemic lesions in the dominant hemisphere exhibited reduced speech activity compared to those with lesions in the non-dominant hemisphere. The degree of cognitive impairment was not associated with the patients' sex (Figure 2.1). No significant differences were observed between the two sexes in terms of cognitive impairment."

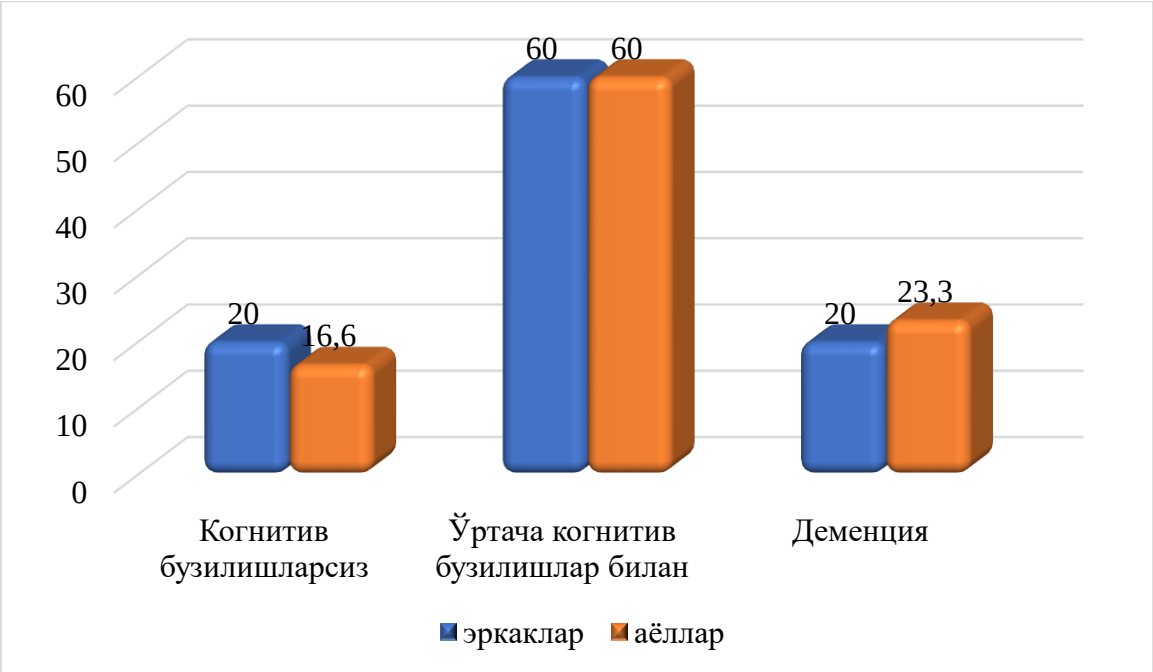



Figure 2.1. Distribution of patients by gender in terms of severity of cognitive impairment (%)

The frequency, degree and side of carotid stenosis did not differ statistically significantly in this group of patients.

"No significant correlation was found between blood pressure levels on the first day of stroke, carotid artery stenosis severity, or NIHSS scores during the acute phase and neuropsychological test outcomes. However, a statistically significant negative correlation was identified between the NIHSS score and clock drawing test results one month after the stroke ($r = –0.35$, $p < 0.05$)."

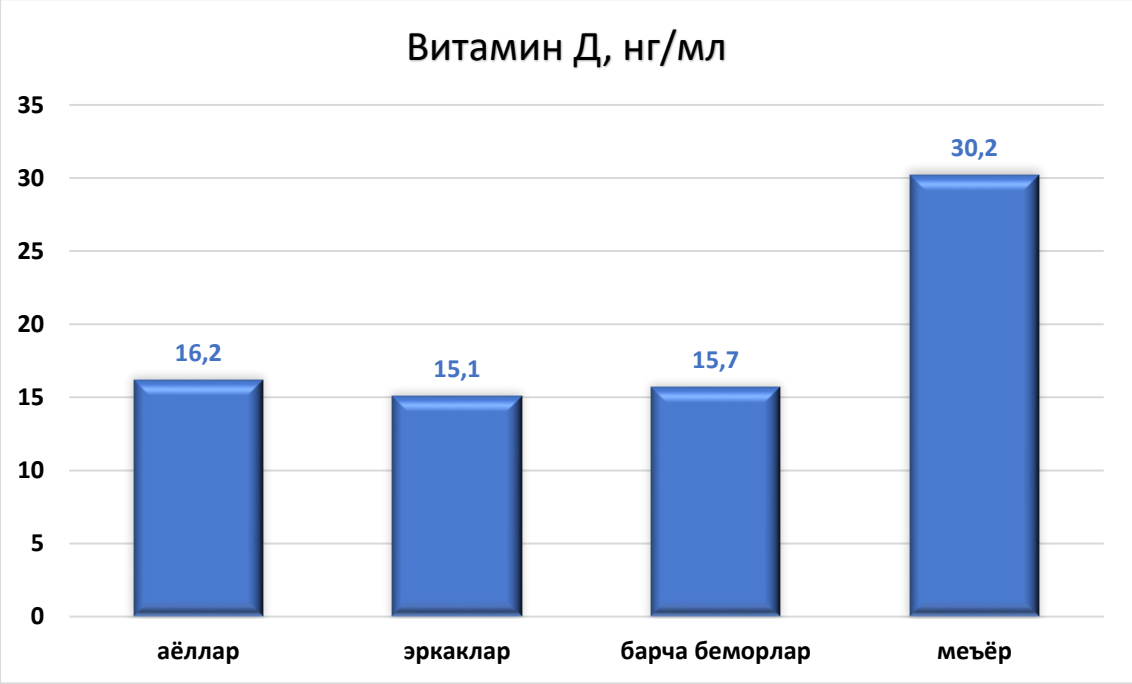
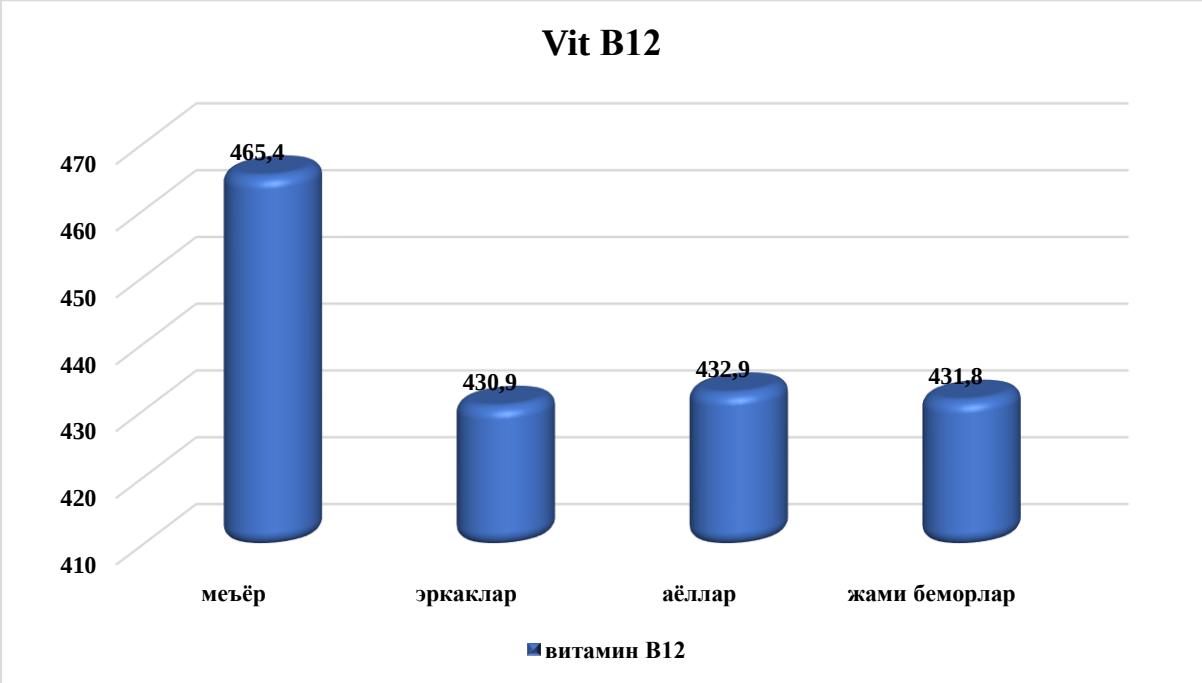



**Figure 2.2. Vitamin D levels in the examined patients**

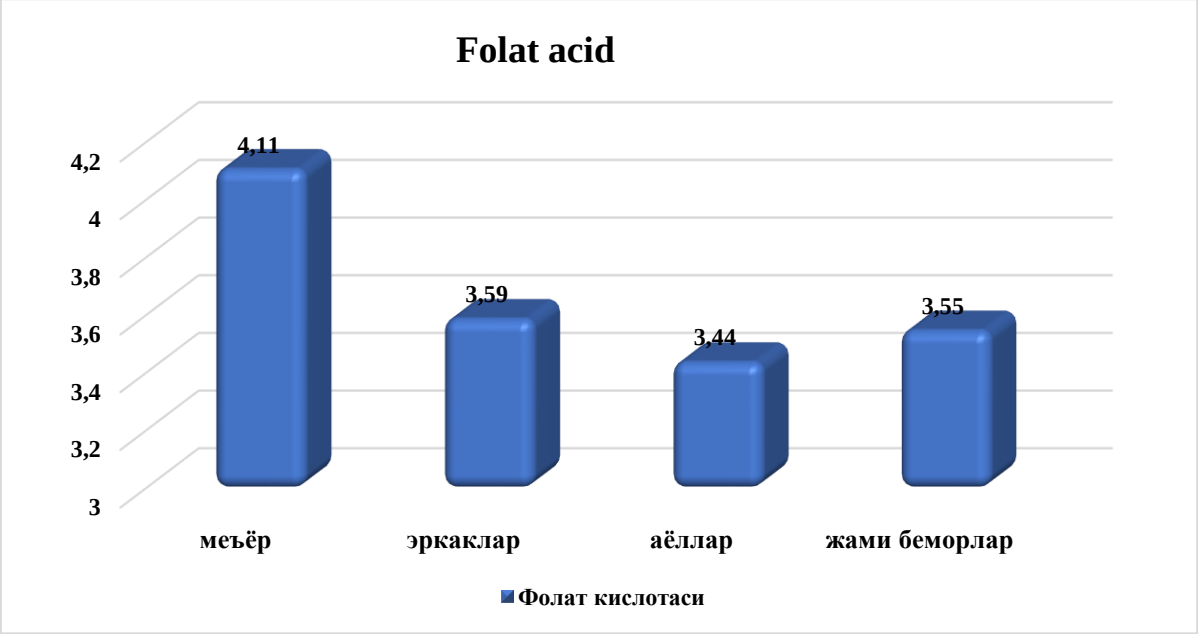
"No statistically significant differences were found between male and female patients in terms of disease stage or laboratory levels of vitamin D, although slightly higher concentrations were noted in women (Figure 2.2). Among the participants, 26.1% exhibited no signs of cognitive decline, 52.9% demonstrated moderate impairment, and 21% met criteria for severe cognitive dysfunction consistent with dementia. The authors' findings support existing evidence linking reduced vitamin D levels to limited sun exposure and inadequate dietary intake [22]. Recent research highlights the important role of vitamin D in maintaining cognitive health. Patients with cognitive deficits often present with markedly lower serum vitamin D levels [24]. Proposed mechanisms for this association include vitamin D's involvement in modulating age-related hippocampal changes, exerting anti-inflammatory effects, and regulating microglial activity [3]. Furthermore, vitamin D has been shown to enhance synaptic plasticity [25] and to elevate glial cell-derived neurotrophic factor (GDNF) levels while restoring tyrosine hydroxylase activity in both the substantia nigra and striatum, thereby supporting dopaminergic neurotransmission (B. Sanchez et al.) [26]."

"Patients who received vitamin D supplementation on a regular basis demonstrated better cognitive performance, supporting the hypothesis that vitamin

D may exert a neuroprotective effect in individuals recovering from stroke. The findings of the present study suggest a potential direct relationship between vitamin D levels and cognitive functioning, with implications for improving patients' quality of life. Additionally, a statistically significant, albeit less pronounced, association was observed between parathyroid hormone (PTH) levels and cognitive outcomes. This may indicate that vitamin D deficiency could concurrently influence both cognitive processes and calcium-phosphorus metabolism. Notably, the correlations identified in our research differ from data reported in several international studies, where elevated PTH levels were significantly and inversely associated with vitamin D deficiency [27]. Such differences may be explained by our study's exclusion of patients with endocrine disorders, which could have influenced hormonal regulation and metabolic interactions."




2.3-picture Validated in patients, the rate of vitamin v12




### 2.4. The amount of folic acid in the examined patients

It is known that cyanocobalamin deficiency, in addition to pernicious anemia, leads to damage to the nervous system in approximately one third of patients. The main neurological manifestations of vitamin B12 deficiency are: damage to the spinal cord (acute combined degeneration of the lateral and dorsal columns or funicular myelosis), brain (dementia), polyneuropathy of the optic nerves and peripheral sensory nerves with the development of neuropathy. Combined damage to the central and peripheral nervous systems is also possible.

"Among the key pathogenic mechanisms of neurological disorders are disruptions in methionine biosynthesis, a deceleration in the oxidation of odd-chain fatty acids, and the subsequent buildup of neurotoxic methylmalonate within the central nervous system. These biochemical disturbances contribute to neuronal fatty degeneration and demyelination of axonal fibers. Furthermore, an accumulation of harmful metabolites in the cerebrospinal fluid (CSF), along with a reduction in neurotrophic support, has been observed, exacerbating neurodegenerative processes."

When studying the amount of vitamin B12 in the examined patients, it was found that there was no significant difference in the level of this vitamin by gender in men and women, but it was 1.05 and 1.1 times lower than the norm, respectively

(Fig. 2.3). According to literature, deficiency of this vitamin can lead to a worsening of cognitive functions in vascular and primary degenerative disorders, as well as to the exacerbation of these disorders.

In patients with cerebral ischemia, vitamin deficiency, in particular folic acid, increases the risk of hyperhomocysteinemia, cardiovascular events and overall mortality, megaloblastic anemia, cognitive disorders, funicular myelosis, polyneuropathy and other neurovascular diseases.

Although the folic acid levels in the examined patients did not differ significantly between the sexes (men – 3.59 + 0.24, women – 3.44 + 0.29), it can be seen that they decreased by 12.6% and 16.3%, respectively, compared to the norm. Compared to the indicators of the general examined patients, they were found to be 2.0% higher in men and 4.0% lower in women (Figure 2.4).

### 2.6. Characteristics of post-stroke cognitive impairment with panic attacks

Women were more likely to experience tremors and nausea during panic attacks.

Atypical symptoms such as a feeling of weakness in the arms or legs; fainting; a feeling of body tilt were not observed in all patients.

Typicality index:

• in men=0.76±0.05

• in women=0.72±0.13

According to the questionnaires, all patients had typical panic attacks.

At the onset of the disease, all patients with panic attacks experienced a fear of death, most often fear of a heart attack and/or stroke; fear of not being able to get help from outside if necessary. Often, women's panic attacks debuted after emotional stress (at the same time, women often spoke about the debut of panic attacks against the background of complete well-being, but a detailed survey revealed an emotionally stressful situation in each of them or a chronic stressful situation occurred). In men, the situation was different: in 40% of men, the onset of panic

attacks occurred the day after a heavy drink. In the remaining 60% of patients, the onset of panic attacks occurred for no apparent reason in the subway, and in more than half of them we were able to determine that they were expecting a significant unpleasant event that day. The clinical characteristics of patients with panic attacks are shown in Tables 3.7 and 3.8.

2.7 **Table**.

**Clinical features of patients with VH.**

| | Men<br>n-20 | Women<br>n-20 |
|---|---|---|
| the average duration of disease (months) | 27 (from 8 till | 31 (from 12 till |
| the frequency of attacks (in months) | 11,4 | 13,2 |
| Night attack (the amount of patients)* | 2 | 1 |
| of a night attack (in months) | 5±2 | 6±1 |
| attack duration (in minutes) | 46,9 | 55,2 |

*- night attack available to all patients from the sensitivitys is.

| | Men<br>n=43 | Women<br>n=37 |
|---|---|---|
| , average duration of disease (months) | 27,2±0,57<br>(from8 till 37) | 31,3±1,62^<br>(from12 till 43) |
| the frequency of attacks (in months) | 11,4±0,88 | 13,2±0.91<br>percentage |
| of the night attack (%)* | 4,7 | 2,7 |
| Night attack (in months) | 5,5±0,37 | 6.5±0,19^ |
| attack duration (minutes) | 46,9±2,72 | 55,2±to 3.45^ |

*- night attack available to all patients from the sensitivitys is.

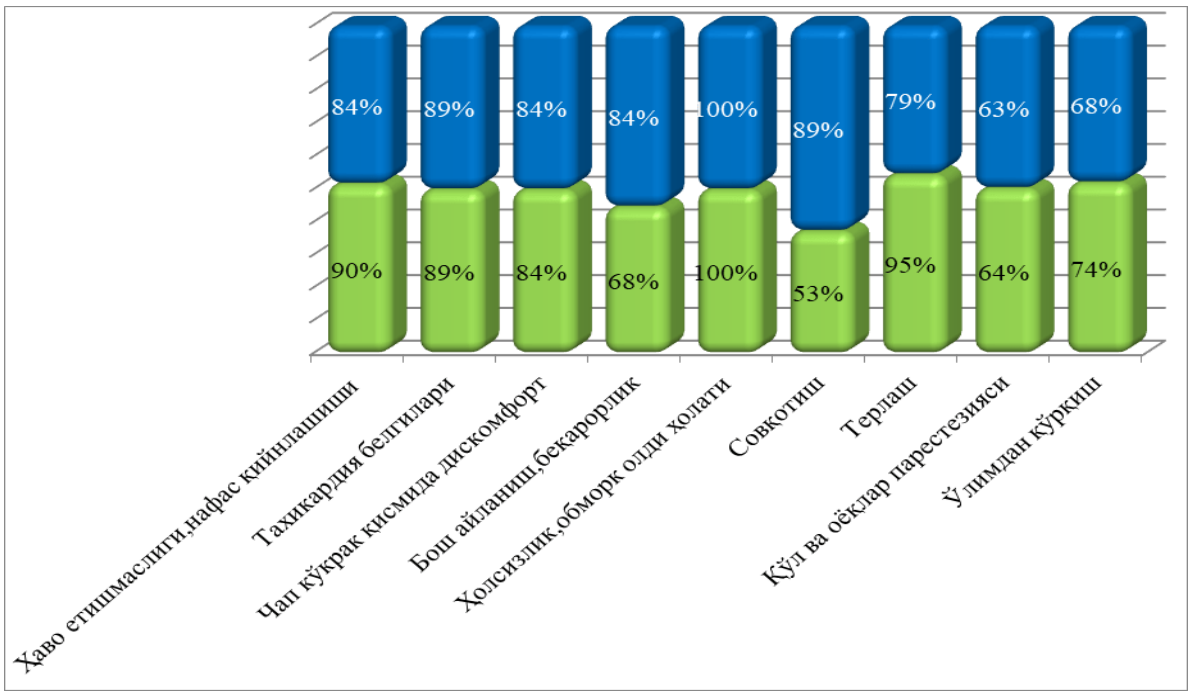
*r<0.05 up to reliable statistical differences in comparison with men

2.8 **Table**.

**Clinical features of a panic attack**

| **Typical symptoms** | Men n=20 | Women n=20 |
|---|---|---|
| the habit of index | 0,76±0.05 up | 0,72±0.13 to |
| the lack of air,a breathing difficulty | 90% | 84% |
| heart beat | 89% | 89% |
| part d on the left chestiskomfort | 84% | 84% |
| to become Home,instability, | 68%, | 84% |
| Weakness,the condition before obmork | 100% | 100% |
| Freezing | 53%* | 89%* |
| hot and cold streams | 79% | 63% |
| Derealizasiya, depersonalizasiya feeling | 74% | 74% |
| Sweating | In 95% | And 79% |
| of the hands and feet paresteziyasi | 64% | 63% |
| from the fear of death and | 74% | 68% |
| shooting inside the feeling of | 100% | 100% |
| Craziness and the action from fear | 42% | 37% |
| ***Atypicalk relieve symptomss*** | | |
| "In the throat; swelling" the feeling of | 47% | 58% |
| weakness in the hands and feet, | 0% | 0% |
| see and hear a decrease of | 37% | 47% |
| walking a violation of | 47% | 42% |
| voice and speech in violation of | 37% | 26% |
| Body belt ekan that feel to make | 0% | 0% |
| draw in the hands and feet | 21% | 16% |
| nausea and vomiting ( | 1%* | 63%* |
| Abdominal diskomfort | 21% | 26% |

* -significant differences between the patient group and the healthy men and women group, $p<0.05$; significant differences between the patient group and the healthy men and women group, $p<0.05$




**2.5 - picture clinical features of panic attack**

All patients with post-stroke cognitive impairment accompanied by panic attacks should be evaluated to ensure that they are in good health and/or have not had a stroke; if necessary, they may be hospitalized. In women, the onset is associated with emotional stress. In men, the situation is different: in 40% of men, the onset of VH was the day after the occurrence of multiple strokes. For the remaining 60%, the onset of VH was observed for no apparent reason in the subway, and more than half of them were expected to have a significant unpleasant event that day (Fig. 23.5).

The following circumstances were responsible for the subsequent onset of panic attacks:

Situational panic attacks that occurred for the first time or in panic situations (for example, in a confined space, when the patient is alone, or when waiting for a significant, usually unpleasant, event).

1. Panic attacks that occur after psychological, physical and mental stress.

2. Panic attacks that develop spontaneously against the background of complete well-being

Simultaneously with post-stroke panic attacks, our patients had low-intensity

attacks (abortive attacks), which were not accompanied by fear and pronounced vegetative disorders, did not adapt the patients to a certain extent and passed independently.

This type of panic attacks was expressed in different patients with different frequency and intensity.

To stop the attacks, men often used beta-blockers, women - valocordin or corvalol in a dose of 25-40 drops. 2 people who regularly took beta-blockers for the treatment of panic attacks used clonazepam to stop their panic attacks.

**2.7. Panic attacks in patients with agorafobiya and social phobia**

Panic qo'zg'atadigan erkaklarning 80% and 75% of women infected with Agorafobiya. Agorafobiya the duration of the disease associated with emas and detected in the initial stages of the disease. Agorafobiyaning according to weight, erkaklar and women almost did not notice. Often from the place of the first attack of a panic attack patients that they can't help anyone when needed or used to escape from a place (2.9-table).

2.9-table

showing a level of verified patien social phobia appearances

| | patients n=40 | | norms n=20 | |
|---|---|---|---|---|
| | men | women | men | women |
| from the situation of fear, the expression considered, | 35±1.27* | 41±2,31 | 25±1,08 | 42±2,24 |
| Situation of the escape expression is considered, | 45±is 2.38 | 50±2,26* | 30±2,13 | 32±2,21 |

*–significant differences between the patient group and the group of healthy men and women, $p<0.05$

Agoraphobia develops significantly more often in men with typical symptoms of panic attacks in the paroxysmal structure. In women, symptoms are of great importance in the development of agoraphobia: swelling of the throat, derealization.

Such an atypical symptom as gait disturbance had a high positive correlation with agoraphobia in both men and women. Gait disturbance always occurred against

the background of non-systemic dizziness, a feeling of general weakness, a state preceding fainting.

When analyzing the degree of expressiveness of fear of various situations, significant differences were observed in men and women, where this condition averaged 35+1.27 and 41.0+2.31 in the corresponding case, while no significant differences were observed between the sexes in the expressiveness of avoidance of situations, i.e., it was 45.0+2.38 and 50.0+2.26 in men and women, respectively.

The severity of fear of situations differed significantly in sick men and healthy men, and did not differ significantly between healthy and sick women. At the same time, the severity of avoidance of situations was significantly more pronounced in sick women than in healthy women and men. Thus, the structure of social phobias in the group of patients is characterized by heterogeneity by sex.

Social phobias were also observed in the group of healthy subjects, but they did not lead to social adaptation.

It is known that panic disorder can also occur as part of social phobia. However, we diagnose panic attacks as part of panic disorder in our patients with agoraphobia. We have come to a similar conclusion based on two clinical observations.

1.Vx analyze the situations that arise in our patients, we never VX our patient in a situation of social importance, for example, we have determined that occur during a public speech. On the contrary, in many cases happened in our patient vx itself; was observed in half of the night; the development of the disease itself or in cases with vx agorafobik happened.

1. Бизнинг беморларимизда ВХ юзага келадиган вазиятларни таҳлил қилиб, биз беморларимизда ВХ ҳеч қачон ижтимоий аҳамиятга эга вазиятда, масалан, оммавий нутқ пайтида содир бўлмаганлигини аниқладик. Аксинча, ВХ кўп ҳолларда бизнинг беморларимизда ўз-ўзидан содир бўлган; ярим тунда кузатилган; касалликнинг ривожланиши билан ВХ ўз-ўзидан ёки Агорафобик ҳолатларда содир бўлган.

2. Бундан ташқари, бизнинг беморларимизнинг ВХ нинг клиник феноменологияси ваҳима бузилишларига хос ВХ мавжудлигини кўрсатди.

Шундай қилиб, бизнинг беморларимизда ВХ тузилишида гипервентилияция синдроми ва нафас қисилиши устунлик қилди, бу ваҳима бузилишлари тузилишида ВХ га хосдир. Ижтимоий фобиялар таркибидаги ВХ учун бошқа аломатлар кўпроқ характерлидир, масалан: юзнинг қизариши, титроқ, кўришнинг пасайиши (Amies et al, 1983; D.J.Stain E.Hollander 2002).

**2.10 table.**

**Data from vegetative questionnaires in a group of healthy subjects and patients with VH.**

| VEGETATION: Objective questionnaire | Bmean emo n=40 | | Norms n=20 | |
|---|---|---|---|---|
| | Men s | Women | Men s | Women |
| 1. Characteristics of changes in skin color and condition<br>- "vascular coral" (spotted hyperemia on the neck, face, chest during external examination)<br>- - the color of the limbs: normal, changed (pale, hyperemic, cyanotic (acrocyanosis), "marble") | 0% | 0% | 0% | 0% |
| 2. Assessment of dermographism:<br>- constant (more than 10 minutes) red, pink, white, high | 10% | 5% | 0% | 0% |
| 3. Evaluation of the level of sweating:<br>- local increase in sweating (exact wetness of palms, feet, armpits, other parts of the body)<br>- - general sweating (increased diffuse moisture of all the above areas, as well as skin in general, chest, back, abdomen, etc.) | 85%* | 75%* | 0% | 30% |
| 4. Presence of temperature changes:<br>- Subfibrillation (constant temperature rise in the range of 37-37.8)<br>- - a sudden rise in temperature in the absence of somatic diseases) | 35% | 35% | 0% | 45%" |
| 5. The presence of health deterioration when the weather changes | 90% * | 70%* | 0% | 0% |
| 6. Poor cold tolerance, congestion | 85%* | 90% | 10% | 90% |

| | | | | |
|---|---|---|---|---|
| 7. Blood pressure lability (in the anamnesis and with bilateral measurement: at the beginning and end of the examination - differences of at least 20-30 mm) | 50%* | 25% | 0% | 0% |
| 8. Heart rate lability (pulse fluctuations at the beginning and end of the test more than 10 beats/min) | 85%* | 15%* | 0% | 40% |
| 9. Presence of hyperventilation syndrome (impaired breathing depth and frequency, feeling of "lack" of air) | of 90%* | 95%* | 0% | 40% |
| 10. Dysfunction of the gastrointestinal tract (in the absence of organic pathology) | of 30% | to 80%** | 20% | 65%* |
| 11. Presence of migraine (tendency to faint) | 10% | 5% | 0% | 0% |
| 12. Acute irritability, anger, indifference, anxiety, fear, sudden mood swings, the presence of anxiety | 100%* | 80% | 15% | 45% |
| 13. Higher neuromuscular excitability:<br>• Xvostek symptom<br>• • * tendency to muscle spasms (carpal cramps - hand, "shortening" of toes, "midwife's" hand, "ballerina's" leg, cramp). | 5% | to 60%* | 0% | 65%* |
| Subjective survey | | | | |
| 1. You (in any time) do you feel batter:<br>- redness of the face<br>- brightness of the face | 75% | 95% | 65% | 80% |
| 2. When do you feel sovqotib oqrish:<br>- Hands and fingers on the feet,<br>- hands and feet Completely, | about 45% | 75% | 70% | 40% |
| 3. Does the color change in your skin (brightness, redness, sprouted to):<br>- Hands and feetof the world on the fingers,<br>- hands and feet Completely | over 50%* | 90% | 70%* | 25% |
| 4. Permanent or excitement with excessive sweating how do you feel (if you answer "yes" is, the word to the bottom 've drawn) | 50% | 100%** | 70%* | 15% |
| 5. You often difficulty, the heart of "extinguished" or "stoppage" a sense of do you have? | 85%* | 75%* | 0% | 25% |
| 6. Do you often have difficulty breathing or a feeling of shortness of breath, rapid breathing? | 85%* | 100%* | 15% | 15% |

| | | | | |
|---|---|---|---|---|
| 7. If gastrointestinal tract function disorder by characterized: abdominal constipation, po-nose, "bloating" lo a batter (the answer is "yes" if, in the word march highlight) | 40% | 90%** | 15% | 55%* |
| 8. Do you have any fainting spells (sudden loss of consciousness or a feeling that you might lose it). If the answer is "yes", specify the conditions: crowded room, excitement, prolonged standing. | 90%* | 75%* | 15% | 15% |
| 9. Do you have paroxysmal headaches (if yes, specify: diffuse or only at the back of the head, constricting or pulsating) | 65% | 70% | 45% | 65% |
| 10. Do you currently feel a decrease in working capacity, rapid fatigue? | 90% | 95% | 70% | 55% |
| 11. Do you experience sleep disturbances? If the answer is "yes", please specify:<br>• Difficulty falling asleep<br>• Light sleep with frequent awakenings<br>• • Insomnia, feeling tired when waking up in the | 70%* | 90%* | 25% | 0% |

* - significant differences between the patient group and the group of healthy men and women, $p<0.05$.

Analysis of data from vegetative questionnaires in the group of healthy and patients with VH (Table 3.10) showed that in the majority of patients of both sexes, the main symptoms were deterioration of health when the weather changed (90% and 70%), poor tolerance to cold, congestion (85% and 90%), the presence of hyperventilation syndrome (90% and 95%) and severe irritability, anger, apathy, feelings of anxiety, fear, sharp mood swings, anxiety (100% and 90%), while the characteristics of changes in skin color and condition, dermagraphism and migraine were absent or expressed at very low levels.

2.11 table

Patients and healthy control group to assess quality of life in

| | The patient men n=20 | patients were female n=20 | healthy men n=10 | healthy women , n=10 |
|---|---|---|---|---|
| physical activity | 29 ±3* | 26 ±3* | 11,1 ±1 | 12 ±2 |
| Present life situation | 28 ±3* | 38±3* | 13 ±2 | 18 ±4 |
| Yourself about thinking | 25 ±3* | 22 ±3 | 12 ±2 | 18 ±5 |
| Future about imagine | 32 ±3* | 28 ±3* | 16 ±2 | 19 ±1 |

Note: * - significant difference between healthy and sick ($p<0.05$)

A comparative analysis of the questionnaires between the examined men and women showed that, while there were no differences in the patients' self-esteem (25+3 and 22+3) and physical activity level (29+3 and 26+3) in assessing their quality of life, we found statistically significant differences in perceptions of the future (32+3 and 28+3) and current life situation (28+3 and 38+3) (Table 3.11).

2.12 table

Assessing quality of life in patients with VH

| | men n=20 | women n=20 |
|---|---|---|
| of a panic attack | 33±2 | 30±2 |
| attack mood and | 30±3 | 32±2 |
| Important people with relationships | 16±2 | 20±3 |
| Social relations of | 17±2* | 23±3 |

* - significant difference between the male and female groups ($p<0.05$)

In patients with panic attacks, differences were mainly found between men and women only in social ties (17+2 and 23+3, respectively) ($p<0.05$).

Thus, the analysis of the questionnaire on the impact of VH on the quality of life revealed a significant decrease in the level of physical activity, dissatisfaction with the current life situation, a decrease in self-esteem and an unfavorable view of the

future. (Tables 2.11, 2.12). In addition, it had a negative effect on both the attitude and the level of social ties of patients with panic attacks, and the latter was much more pronounced in sick women than in men.

## III Chapter.

# PANIC ATTACKS ACCOMPANIED WITH POST-STROKE COGNITIVE DISORDERS WAYS TO INCREASE THE EFFECTIVENESS OF THE TREATMENT OF WORKERS

## 3.1. Results of clinical and psychophysiological analysis of patients' condition in treatment dynamics

By the end of the second month of fluoxetine treatment, 90% of patients (17 men and 18 women) reported cessation of VH. Panic attacks continued in 3 men and 2 women (one woman had not only daytime but also nocturnal panic attacks), but their frequency and intensity decreased. A woman who had nocturnal panic attacks, after a course of fluoxetine treatment, the number of attacks decreased by half, but they did not completely regress. In men, after treatment, nocturnal attacks of panic attacks completely regressed. All patients noted good tolerance of the drug, in the first week of taking the drug in 2 men and 2 women, non-systemic dizziness increased, which decreased by the end of the first week of taking the drug, but did not completely regress. These patients were characterized by the onset of vertigo.

Agoraphobia regressed in 95% of women and 100% of men. Patients stopped fleeing from places that previously caused agoraphobic reactions (metro, bus, elevator, loneliness, etc.). They had more freedom of movement and less dependence on loved ones. Agoraphobia persisted in one woman who had both panic attacks (night and day) at the same time.

3.1 table.

Patients in the group of giperventilyatsiya symptoms of frequency and intensitychanges in the dynamics of treatment

| Men n=20 | | | | Women n=20 | | | |
|---|---|---|---|---|---|---|---|
| the frequency of | | Intensivesocial | | Frequency | | Intensivemore | |
| before cure | Cure after | cure before | Cure after | cure before | Cure after | Cure before | the treatment and after the |
| 17,56±1,24 | 6,9±1.3 indicator | 22,7±1,1 | 9,7±1,01 | 20,3±1,28 | 8,4±0.5 to | 20,5±1,5* | 9,1±1,09 |

* - significant differences between the groups of patients before and after treatment of men and women, $p<0.05$

When analyzing the dynamics of changes in the frequency and intensity of hyperventilation symptoms in the group of patients, statistically significant differences were found between men and women, and on average, the frequency of hyperventilation before and after treatment was 17.56±1.24 and 6.9±1.3 in men, respectively, and 20.3±1.28 and 8.4±0.5 in women. The intensity of hyperventilation in patients was 22.7±1.1 and 9.7±1.01, 20.5±1.5 and 20.5±1.5, respectively ($p<0.05$) (Table 3.1).

Thus, against the background of ongoing therapy, the frequency and intensity of hyperventilation syndrome in both men and women decreased significantly.

Thus, against the background of ongoing therapy:

1. Both men and women began to pay significantly less attention to the following symptoms: decreased performance; fatigue; paroxysmal headaches; shortness of breath or feeling of lack of air; rapid breathing; anxiety; irritability; feelings of anger, anxiety; fear; sharp mood swings; asthenia; disorders: sleep; the presence of hyperventilation syndrome.

3.2 table.

The results of inquiries and vegetative cure before and after

| VEGETATIKA: Lens survey | cure before n=20 | | Cure after n=20 | |
|---|---|---|---|---|
| | Men s | Women | Men s | Women |
| 1. Characteristics of changes in skin color and condition<br>- "vascular coral" (spotted hyperemia on the neck, face, chest during external examination)<br>- the color of the limbs: normal, changed (pale, hyperemic, cyanotic (acrocyanosis), "marble") | 0% | 0% | 0% | 0% |
| 2. Assessment of dermographism:<br>- constant (more than 10 minutes) red, pink, white, high | 10% | 5% | 10% | 5% |
| 3. Assessment of dermographism:<br>- constant (more than 10 minutes) red, pink, white, high<br>- general sweating (increased diffuse moisture of all the above areas, as well as the skin, chest, back, abdomen, etc.) | 85% | 75% | 80% | 75% |
| 4. Do you experience excessive sweating, either constantly or with excitement (if the answer is "yes", underline the word) | 35% | 35% | 15% | 15% |
| 5. Do you often feel dizzy, your heart is "fading" or "stopping"? | 90% | 70% | 75% | 70% |
| 6. Do you often have difficulty breathing or a feeling of lack of air, rapid breathing | 85% | 90% | 80% | 85% |
| 7. If the gastrointestinal tract is characterized by impaired function | by 50%* | 25% | 10% | 25% |
| 8. Do you have fainting spells | 85% | 15% | 15%* | 10% |
| 9. Do you have paroxysmal headaches | 90%* | 95%* | 0% | 5% |
| 10. Do you currently feel a decrease in working capacity, rapid fatigue | 30% | 80% | 35% | 75% |
| 11. Do you experience sleep disturbances? | 10% | 5% | 5% | 5% |

* - significant differences between the groups of patients before and after treatment of men and women, $p<0.05$

2. In addition, men began to experience significantly less: instability of blood pressure, lability of heart rate, feeling of heaviness, "fading" or "stopping" of the

heart (Table 3.2).

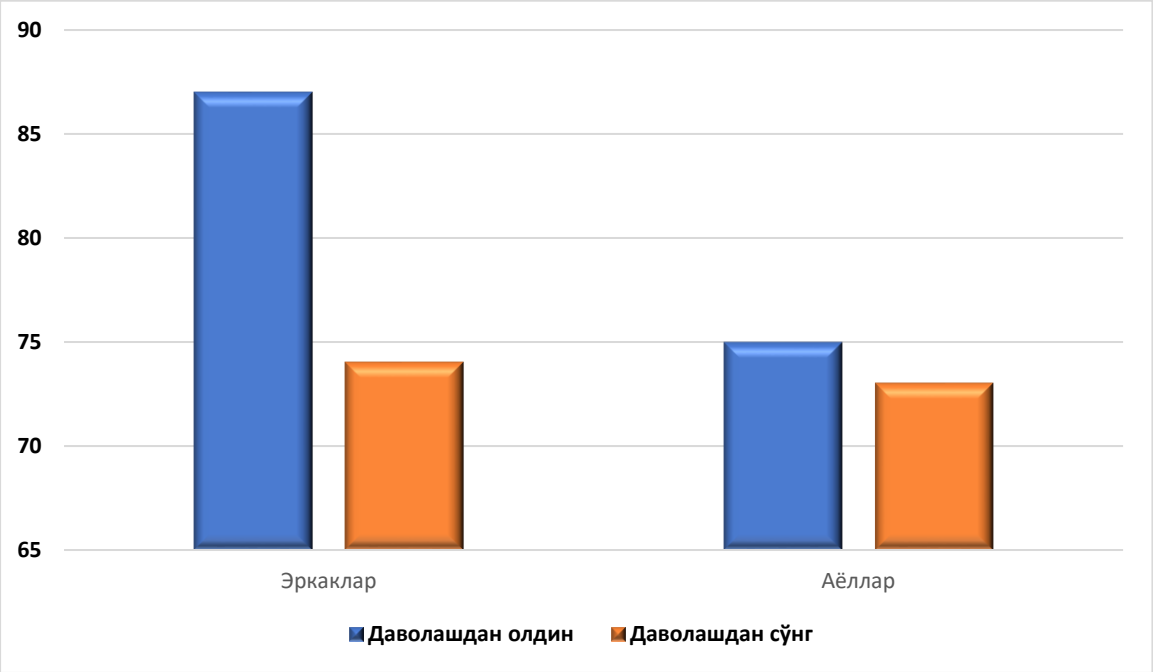



Fig. 3.1. Heart rate before and after treatment

Thus, after the course of treatment, the heart rate in sick men decreased significantly. No dynamics were observed in sick women, since even before treatment their heart rate indicators were within the normal range and did not differ from healthy subjects (Fig. 3.1)

Table 3.3

Assessment of quality of life in patients and healthy groups

| | Males before treatment: n=20 | Female before treatment | Males after treatment n=20 | Females after treatment n=20 |
|---|---|---|---|---|
| physical strain | 29±3 | 26±3 | 22±1* | 21,6±3* |
| Current life situation | 28±3 | 38±3 | 19±3* | 19±2* |
| thinking about yourself | 25±3 | 22±3 | 20±1 | 20±2* |
| the future about imagine | 32±3 | 28±3 | 22±5* | 21±3* |

* - significant differences between the groups of patients before and after treatment of men and women, $p<0.05$

Thus; after treatment, the quality of life in all “measurable” parameters

improved significantly: physical activity increased, self-esteem improved, men and women had a more favorable view of the future and current life situation (Table 3.3).

6 months after the end of therapy, 47% of men (8 people) and 50% of women (9 people) completely recovered from the attacks during VH therapy, the attacks resumed. The frequency and intensity of these attacks were significantly lower than before treatment, nocturnal attacks did not occur. In most patients (78% of women and 38% of men), decompensation occurred against the background of a stroke and was associated with concern for the health and life of their loved ones. In 50% of men, decompensation occurred against the background of problems related to their professional activities, in 12% of women and in 12% of men - spontaneously.

A retrospective analysis of these patients showed that, according to the Spielberger questionnaires, they had a significantly higher level of personal and reactive anxiety before treatment, and a significantly higher level of personal anxiety after treatment (see Tables 3.4, 3.5).

Table 3.4

The level of anxiety in patients with relapsed VH and VH before treatment according to the Spielberger anxiety questionnaire

| | VX regressi | | regress vx no | |
|---|---|---|---|---|
| | Men, n=8 | Women n=9 | Males, n=9 | Women n=9 |
| Personal concern | 40,6±2.8* | 42,5±4.5* | 45,4±1.7 was | 50,8±3,9 |
| Reactive anxiety | 44,2±3,b* | 45,1±2.6* | 49,5±2.1 | 52,4±4.1 |

*- significant difference between groups ($p<0.05$)

In patients with relapsed VH according to the Spielberger Anxiety Questionnaire, the level of personal and reactive anxiety was more likely to be associated with the presence of panic attacks among women. The level of personal anxiety was 42.5+2.8 units in patients with relapsed VH, and 50.8+3.9 units in those without relapsed VH, while the level of reactive anxiety was 45.1+2.6 and 52.4+4.1 units, respectively ($p<0.05$).

Table 3.5

The level of anxiety in patients after treatment with relapsed VH and the Spielberger Anxiety Questionnaire in patients with VH

| | Regressli VX | | VX we regress | |
|---|---|---|---|---|
| | Men n-8 | Women n-9 | Men n-9 | Women n=9 |
| Personal | 36,4±2.1* | 37.8±2,4* | 40.3±1.5 | 41.2±1.7 |
| Reactive | 40,9±1,4 | 42,3±1.5 | 42,1±1.7 | 40,7±1.3 |

*- significant difference between groups ($p<0.05$)

Thus, high levels of reactive and subjective anxiety predict poor long-term (more than 6 months) results of fluoxetine treatment. In this regard, patients with a high level of anxiety are recommended to undergo a longer course of maintenance therapy with SSRIs (more than 4 months) after the relief of VD.

In 3 men and 2 women, who did not have complete regression of VD symptoms during therapy, the frequency and intensity of VD remained at the level achieved after treatment. These patients periodically underwent courses of treatment with psychotropic drugs of other groups, since the SSRI group caused them severe dizziness at the initial stage of treatment. These observations indicate that patients in whom dizziness is one of the dominant symptoms in the structure of VD require the selection of antidepressants from other pharmacological groups.

### 3.2. Characteristics of patients with post-stroke cognitive impairment without panic attacks.

In our group 1 patients, it was found that post-stroke cognitive impairment was accompanied by post-stroke depression.

Analysis of the localization of blood vessels showed that in 50% of patients, the BMSD occurred in the right hemisphere of the brain and in 50% of patients in the left hemisphere. The analysis showed that the occurrence of depression was not associated with the localization of the stroke (Fig. 3.2).

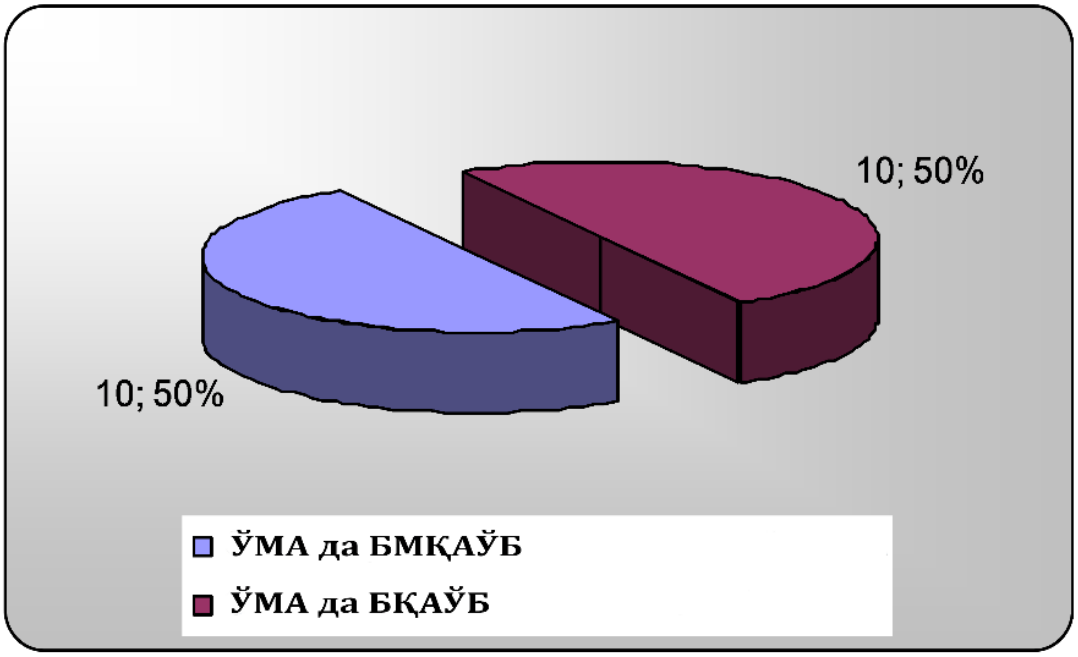



Figure 3.2. Analysis of stroke localization.

Analysis of the study of post-stroke depression (PSD) showed that 60% of patients examined after 2 weeks of stroke developed depression. The depressive component was a minor depressive episode in 20% of patients and a major depressive episode in 40% of patients (Figure 3.3).

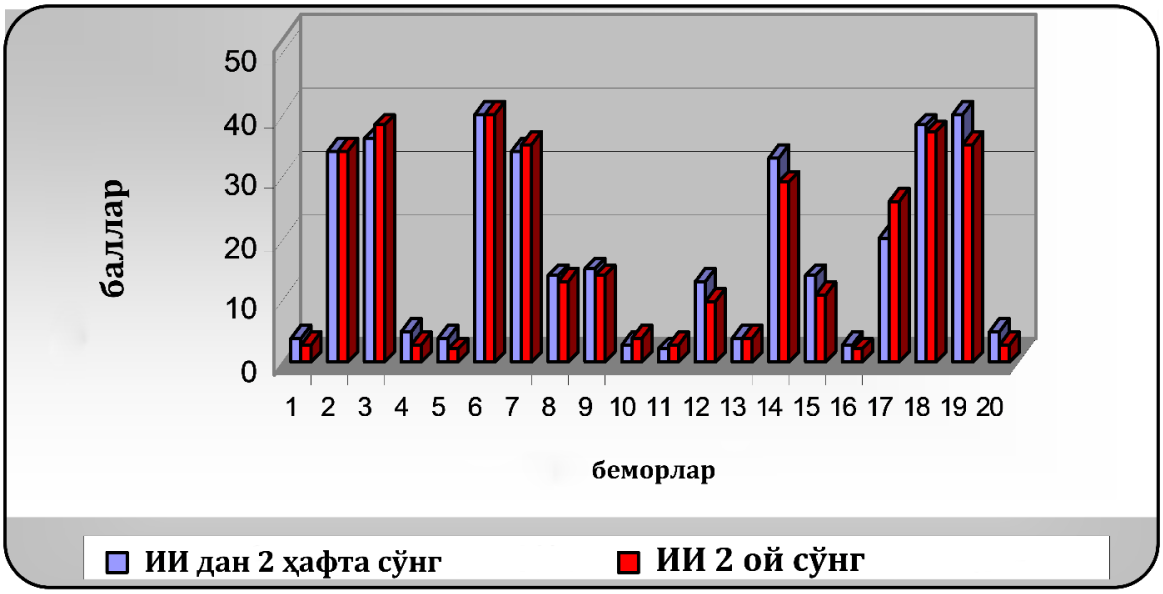



Figure 3.3. Assessment of VH according to the Hamilton scale 2 weeks after the II.

Assessment of VH after two months showed that in 25% of patients the depressive state worsened in the form of an increase in the sum of the Hamilton depression scale scores by an average of 2.25±6.75 points. At the same time, in 15% of patients the sum of the DE assessment scores remained at the same level, and in the remaining patients with a diagnosis of DE, a decrease in depression in the sum of the scores was observed (average 2.25±15.75) (Figure 3.3 and Table 3.6).

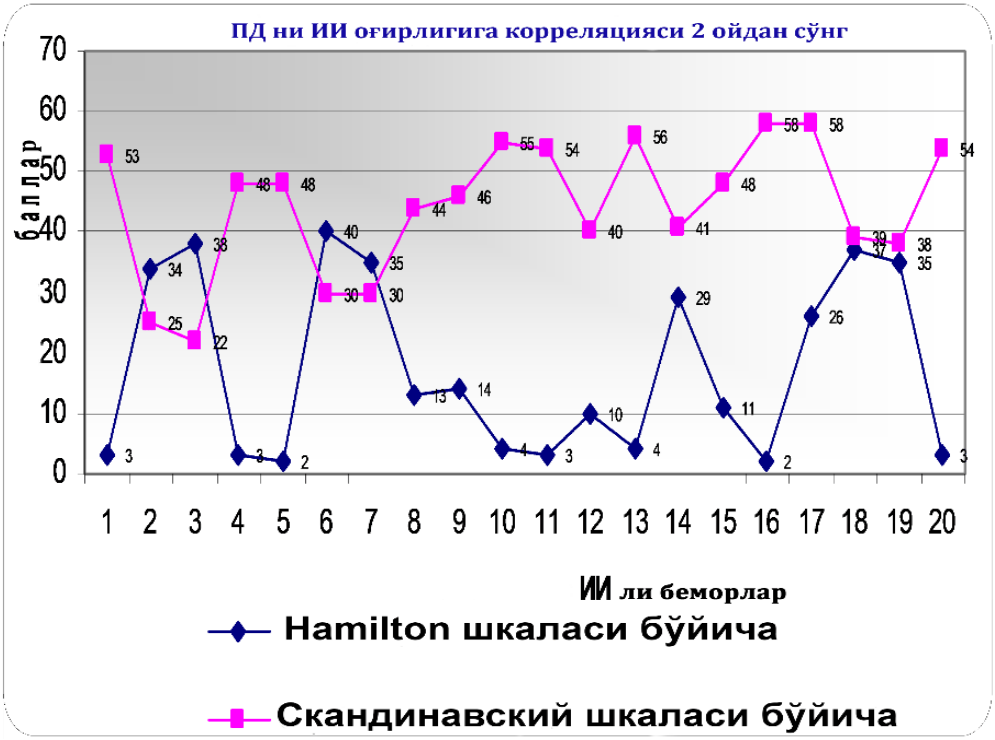
Table 3.6

Assessment of VH according to the Hamilton scale.

| **The number of patients** | **VX treatment** | | **VX at the time of treatment and after treatment** | | |
|---|---|---|---|---|---|
| | **2 weeks** | **2 months** | **3 months** | **4 months** | **6 months** |
| 1 | 4 | 3 | 3 | 2 | 2 |
| 2 | 34 | 34 | 33 | 23 | 20 |
| 3 | 36 | 38 | 36 | 24 | 21 |
| 4 | 5 | 3 | 3 | 3 | 3 |
| 5 | 4 | 2 | 2 | 4 | 3 |
| 6 | 40 | 40 | 39 | 29 | 15 |
| 7 | 34 | 35 | 33 | 20 | 14 |
| 8 | 14 | 13 | 12 | 8 | 6 |
| 9 | 15 | 14 | 12 | 7 | 5 |
| 10 | 3 | 4 | 3 | 2 | 3 |
| 11 | 2 | 3 | 2 | 1 | 1 |
| 12 | 13 | 10 | 9 | 7 | 5 |
| 13 | 4 | 4 | 3 | 2 | 1 |
| 14 | 33 | 29 | 26 | 15 | 8 |
| 15 | 14 | 11 | 9 | 7 | 5 |
| 16 | 3 | 2 | 2 | 2 | 1 |
| 17 | 20 | 26 | 25 | 20 | 15 |
| 18 | 38 | 37 | 29 | 15 | 13 |
| 19 | 40 | 35 | 34 | 33 | 25 |
| 20 | 5 | 3 | 2 | 1 | 2 |
| | ОДЭ - 8<br>МДЭ - 4 | ОДЭ-8<br>МДЭ-4 | ОДЭ-8<br>МДЭ-4 | ОДЭ-8<br>МДЭ-6 | ОДЭ-12<br>МДЭ-5 |

| | | | | | |
|---|---|---|---|---|---|
| | БДЭ- 8 | БДЭ-8 | БДЭ-8 | БДЭ- 6 | БДЭ-3 |

Then, we made a correlation between II and VX weight within 6 months after stroke (Table 3.7), which revealed certain results. The analysis showed that the severity of II and VX has a direct relationship. Figure 3.4 shows that the more severe the stroke and the more accurately recorded VX. And vice versa, the milder the




stroke and the milder the VX. (Figure 3.4).clearly recorded ebuilt in the us. And on the contrary, stroke how light is , and VX is so light that. (3.4-picture).

Figure 3.4. Correlation between severity of stroke and course of VH after 2 months.

All patients were prescribed the antidepressant Fluoxetine 20 mg at a dose of 20 mg. Each 1 capsule 1 time per day for 2 months. Analysis of the dynamics of treatment was carried out for 2,4,6 months. It can be seen from the figure that treatment of all patients began 2 months after stroke. The results of the study showed that after 4 months in patients with VH, the depressive episode disappeared with a decrease or regression of neurological deficit or turned from a major depressive episode to a minor depressive episode with a decrease in the Hamilton depression score (Figure

3.5).

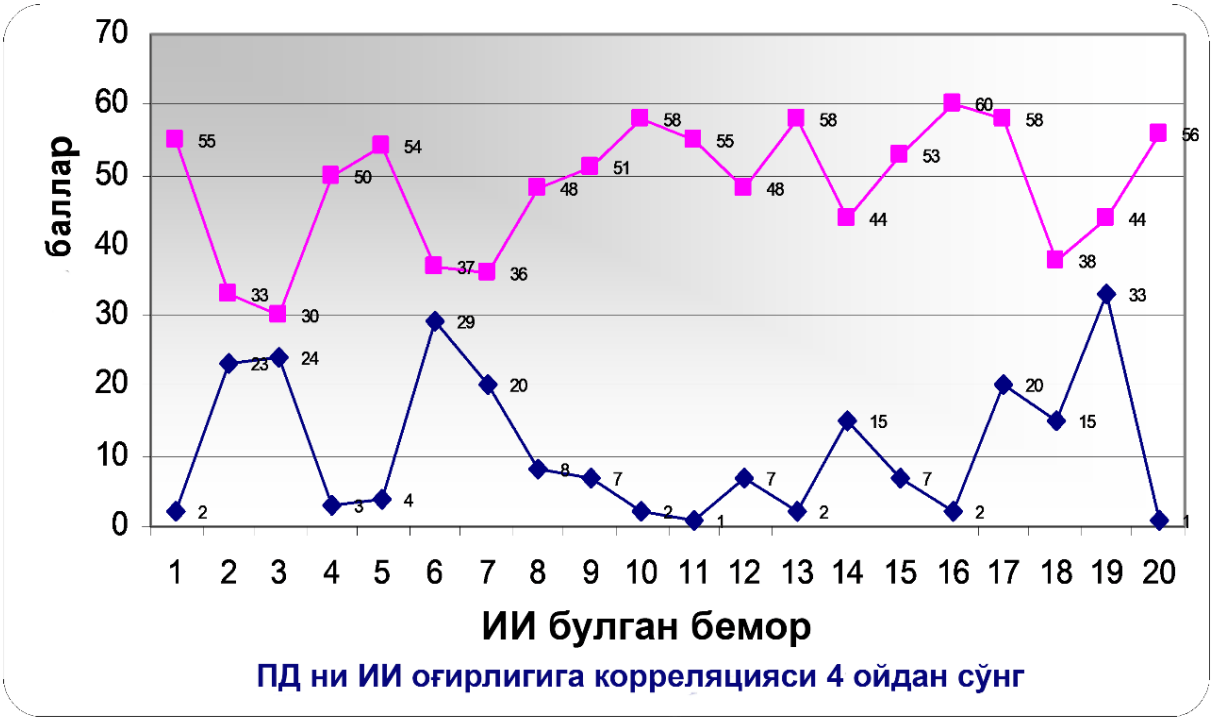



Figure 3.5. Correlation of II severity and VX progression after 4 months.

Table 3.7

Correlation of II severity and VX progression after 6 months.

| The number of patients | Evaluation according to the VX Hamilton scale | Assessment of VX severity according to the Scandinavian scale | Hamilton scale of VXto evaluate | Assessment of stroke severity according to the Scandinavian scale | Hamilton scale of VXto evaluate | Assessment of stroke severity according to the Scandinavian scale |
|---|---|---|---|---|---|---|
| | II 2 months after | | II 4 months after | | II after 6 months from | |
| 1 | 3 | 53 | 2 | 55 | 2 | 58 |
| 2 | 34 | 25 | 23 | 33 | 20 | 38 |
| 3 | 38 | 22 | 24 | 30 | 21 | 33 |
| 4 | 3 | 48 | 3 | 50 | 3 | 56 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 5 | 2 | 48 | 4 | 54 | 3 | 55 |
| 6 | 40 | 30 | 29 | 37 | 15 | 40 |
| 7 | 35 | 30 | 20 | 36 | 14 | 39 |
| 8 | 13 | 44 | 8 | 48 | 6 | 51 |
| 9 | 14 | 46 | 7 | 51 | 5 | 53 |
| 10 | 4 | 55 | 2 | 58 | 3 | 58 |
| 11 | 3 | 54 | 1 | 55 | 1 | 55 |
| 12 | 10 | 40 | 7 | 48 | 5 | 51 |
| 13 | 4 | 56 | 2 | 58 | 1 | 58 |
| 14 | 29 | 41 | 15 | 44 | 8 | 46 |
| 15 | 11 | 48 | 7 | 53 | 5 | 53 |
| 16 | 2 | 58 | 2 | 60 | 1 | 60 |
| 17 | 26 | 58 | 20 | 58 | 15 | 58 |
| 18 | 37 | 39 | 15 | 38 | 13 | 42 |
| 19 | 35 | 38 | 33 | 44 | 25 | 44 |
| 20 | 3 | 54 | 1 | 56 | 2 | 56 |

With standard treatment for 6 months of patients with IBS, 40% of patients showed significant improvement, 30% showed moderate improvement, and 30% showed mild improvement. The Scandinavian scale, in which the assessment of the VH after 6 months showed continued ED, but was manifested by a transition from CDE to Minor ED and a decrease in the estimated CDE score on the Hamilton scale (Fig. 3.6).

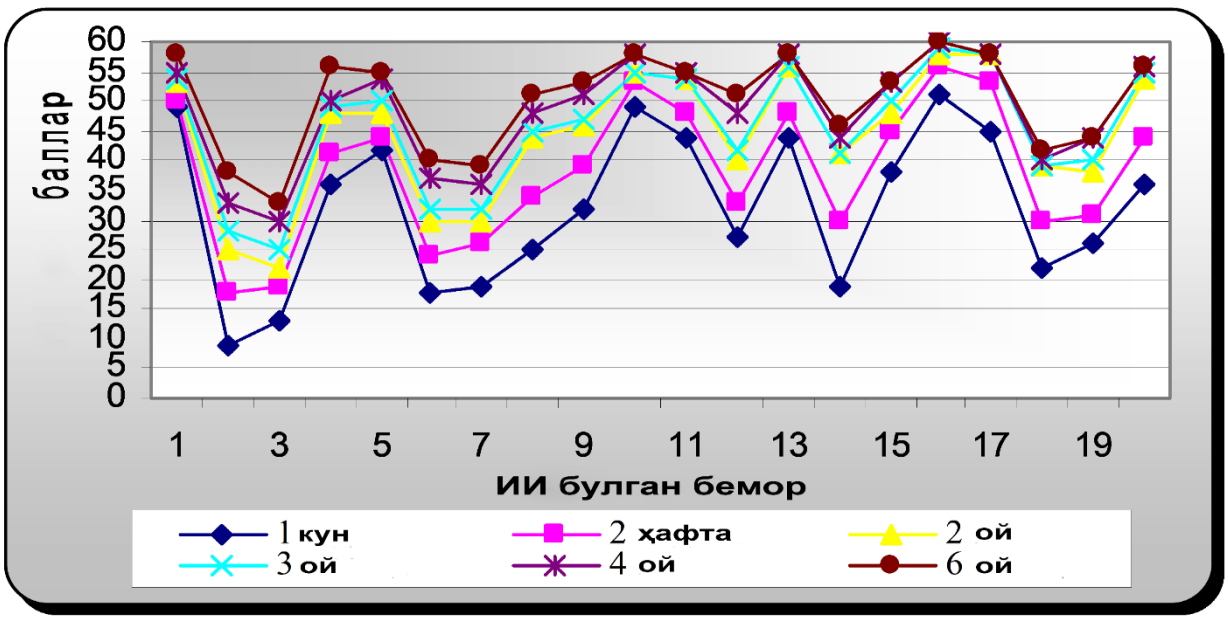
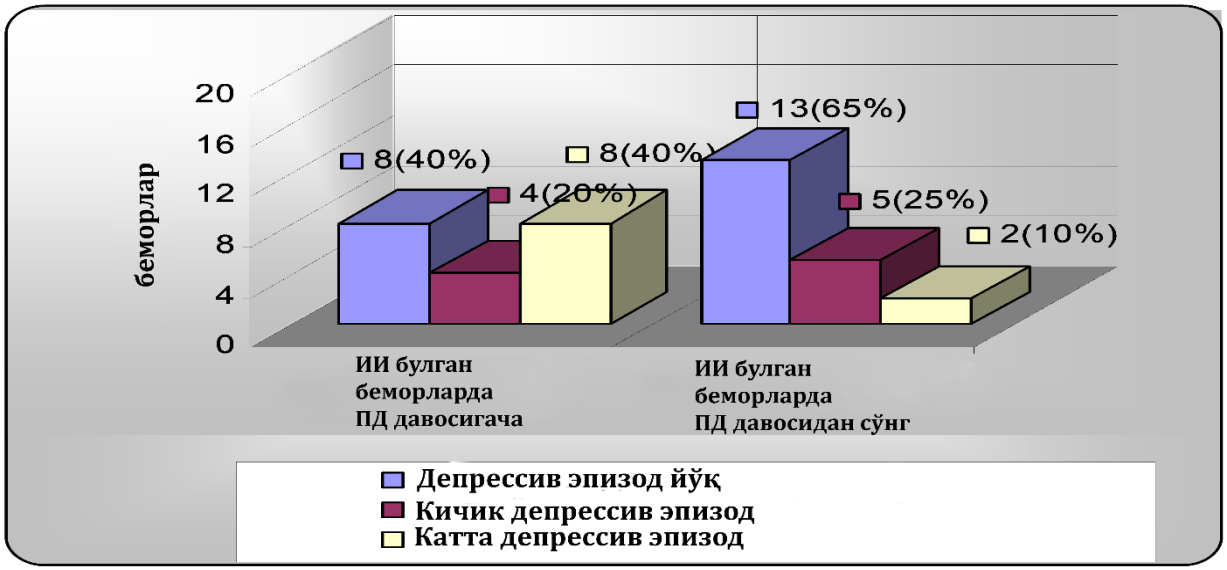


Figure 3.6. Assessment of stroke severity according to the Scandinavian scale during 6 months of standard treatment.

In patients with IHD, 20% of patients had mild ED and 40% had CDE before treatment with antidepressants (ADP) at the second month. After 3 months of treatment with fluoxetine 20 mg, CDE was converted to mild ED in 6% of patients, and 10% remained CDE, but with a decrease in the percentage of assessments to 37.1% (Figure 3.7).




3.7. picture Dynamics of depressive episodes in examined patients.

## CONCLUSION

Based on the results of the dissertation work on the topic "Cognitive disorders accompanied by panic attacks in stroke patients", the following conclusions were drawn.

1. In a patient in the acute phase of ischemic stroke, mild hemiparesis was detected with a decrease in muscle strength up to 4 points in 73% of patients, moderate hemiparesis was detected in 27% of patients, mainly movement disorders were observed in the hand, mainly in its proximal part, speech disorders were detected in 44% of cases, of which 32% had dysarthria and 12% had mild motor aphasia.

2 "The studied cohort exhibited a high frequency of comorbid conditions, including agoraphobia, social phobia, and panic disorder in conjunction with depressive symptoms. Gender-specific and temporal patterns were noted: in male patients, agoraphobia more commonly emerged as a consequence of secondary depressive states, whereas in female patients, social phobia characterized by avoidance of socially relevant situations tended to evolve into agoraphobia."

3. Attention disorders were observed in 85% of patients with moderate cognitive impairment, visual memory disorders in 66.7% of patients, auditory-speech memory disorders in 77.3%, visual-spatial disorders in 47% of patients, and speech disorders in 91% of patients. Attention disorders, auditory-speech memory, and speech disorders were detected in 100% of patients with dementia, visual memory disorders in 91% of patients, and visual-spatial disorders in 73% of patients.

4. After 6 months of complex therapy, panic attacks completely disappeared in 47% of men and 50% of women. In most patients (78% of women and 38% of men), decompensation occurred against the background of a stroke and was associated with concerns for the health and life of their loved ones, in 50% of men, decompensation occurred against the background of problems related to their professional activities, in 12% of women and 12% of men - spontaneously.

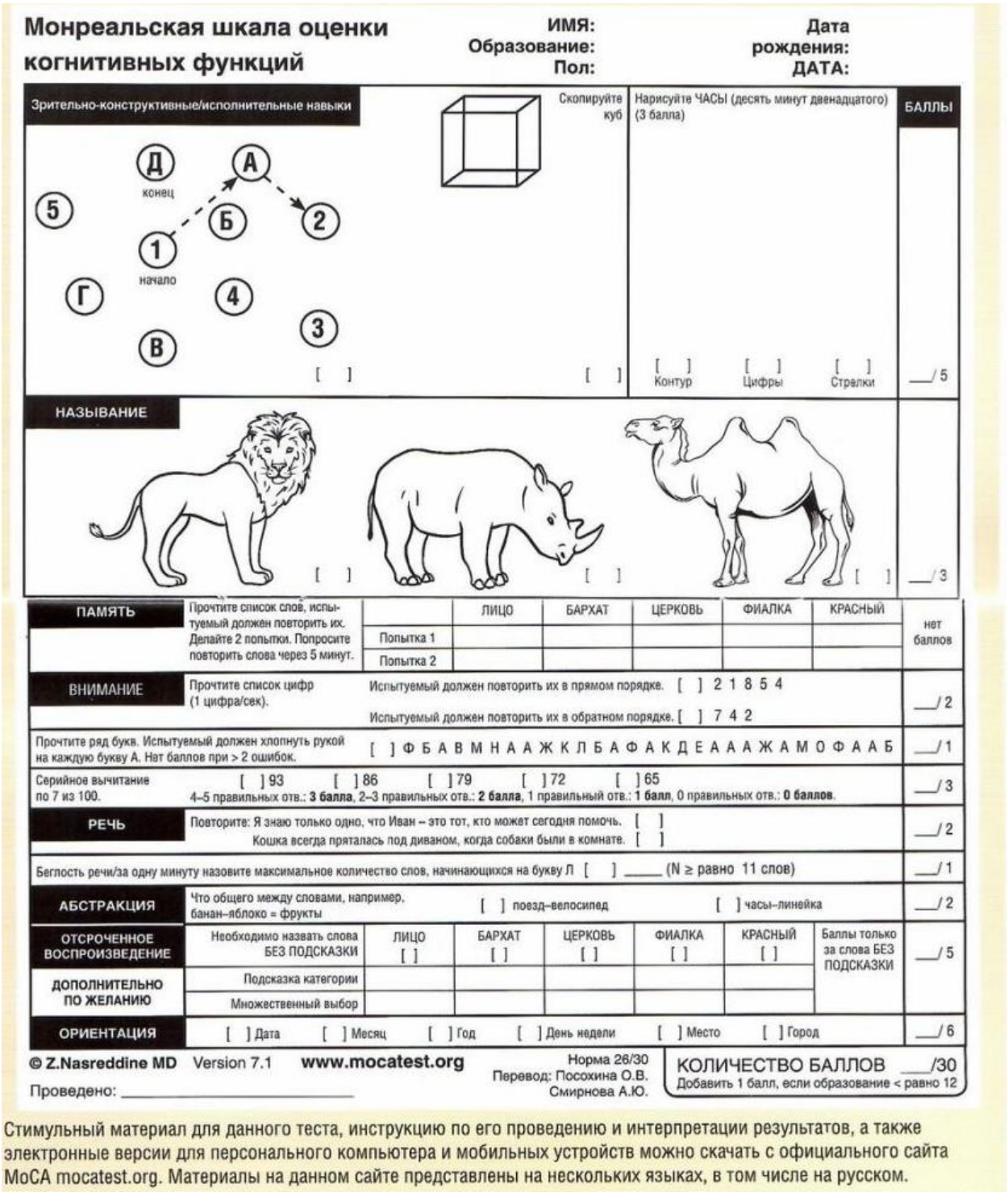
5. "Among patients with ischemic stroke, a six-month course of standard

therapy resulted in marked clinical improvement in 40% of cases, moderate improvement in 30%, and slight improvement in the remaining 30%. Evaluation of psychoemotional status using the Scandinavian scale indicated persistence of depressive symptoms; however, a shift from clinically defined depression (CDE) to milder depressive episodes (Minor DE) was observed, accompanied by a reduction in Hamilton depression scale scores."1-app.

**1-app**

## MoSA-Montreal Cognitive Scale.

## Questionnaire for the assessment of cognitive functions.



**Монреальская шкала оценки когнитивных функций**

ИМЯ: Образование: Пол:

Дата рождения: ДАТА:

**Зрительно-конструктивные/исполнительные навыки** — БАЛЛЫ

Скопируйте куб

Нарисуйте ЧАСЫ (десять минут двенадцатого) (3 балла)

Д (конец), А, 5, Б, 2, 1 (начало), Г, 4, 3, В

[ ] [ ] [ ] Контур [ ] Цифры [ ] Стрелки ___/5

**НАЗЫВАНИЕ**

[ ] [ ] [ ] ___/3

**ПАМЯТЬ** Прочтите список слов, испытуемый должен повторить их. Делайте 2 попытки. Попросите повторить слова через 5 минут.

| | ЛИЦО | БАРХАТ | ЦЕРКОВЬ | ФИАЛКА | КРАСНЫЙ | нет баллов |
|---|---|---|---|---|---|---|
| Попытка 1 | | | | | | |
| Попытка 2 | | | | | | |

**ВНИМАНИЕ** Прочтите список цифр (1 цифра/сек).

Испытуемый должен повторить их в прямом порядке. [ ] 2 1 8 5 4

Испытуемый должен повторить их в обратном порядке. [ ] 7 4 2 ___/2

Прочтите ряд букв. Испытуемый должен хлопнуть рукой на каждую букву А. Нет баллов при > 2 ошибок. [ ] Ф Б А В М Н А А Ж К Л Б А Ф А К Д Е А А А Ж А М О Ф А А Б ___/1

Серийное вычитание по 7 из 100. [ ] 93 [ ] 86 [ ] 79 [ ] 72 [ ] 65

4–5 правильных отв.: **3 балла**, 2–3 правильных отв.: **2 балла**, 1 правильный отв.: **1 балл**, 0 правильных отв.: **0 баллов**. ___/3

**РЕЧЬ** Повторите: Я знаю только одно, что Иван – это тот, кто может сегодня помочь. [ ]

Кошка всегда пряталась под диваном, когда собаки были в комнате. [ ] ___/2

Беглость речи/за одну минуту назовите максимальное количество слов, начинающихся на букву Л [ ] ____ (N ≥ равно 11 слов) ___/1

**АБСТРАКЦИЯ** Что общего между словами, например, банан–яблоко = фрукты [ ] поезд–велосипед [ ] часы–линейка ___/2

| | Необходимо назвать слова БЕЗ ПОДСКАЗКИ | ЛИЦО [ ] | БАРХАТ [ ] | ЦЕРКОВЬ [ ] | ФИАЛКА [ ] | КРАСНЫЙ [ ] | Баллы только за слова БЕЗ ПОДСКАЗКИ |
|---|---|---|---|---|---|---|---|
| **ОТСРОЧЕННОЕ ВОСПРОИЗВЕДЕНИЕ** | | | | | | | ___/5 |
| **ДОПОЛНИТЕЛЬНО ПО ЖЕЛАНИЮ** | Подсказка категории | | | | | | |
| | Множественный выбор | | | | | | |

**ОРИЕНТАЦИЯ** [ ] Дата [ ] Месяц [ ] Год [ ] День недели [ ] Место [ ] Город ___/6

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Норма 26/30
Перевод: Посохина О.В.
Смирнова А.Ю.

КОЛИЧЕСТВО БАЛЛОВ ___/30
Добавить 1 балл, если образование < равно 12

Проведено: _____

Стимульный материал для данного теста, инструкцию по его проведению и интерпретации результатов, а также электронные версии для персонального компьютера и мобильных устройств можно скачать с официального сайта MoCA mocatest.org. Материалы на данном сайте представлены на нескольких языках, в том числе на русском.

**2-app**

## Khachinsky scale

| | there is | no |
|---|---|---|
| sudden start, | 2 | 0 |
| - stage progression | 1 | 0 |
| fluctuation period | 2 | 0 |
| night terrors | 1 | 0 |
| the relative preservation of the person, | 1 | 0 |
| depression, | 1 | 0 |
| somatic complaints, | 1 | 0 |
| emotional lability | 1 | 0 |
| hypertension, | 1 | 0 |
| of stroke in anamnesis | 2 | 0 |
| atherosclerosis, | 1 | 0 |
| pathological focal signs | 2 | 0 |
| focal neurological signs | 2 | 0 |

Assessment:

>7–dementsiyasi blood vessels

5-6–mixed type (can be)

0-4–alzheimer's disease

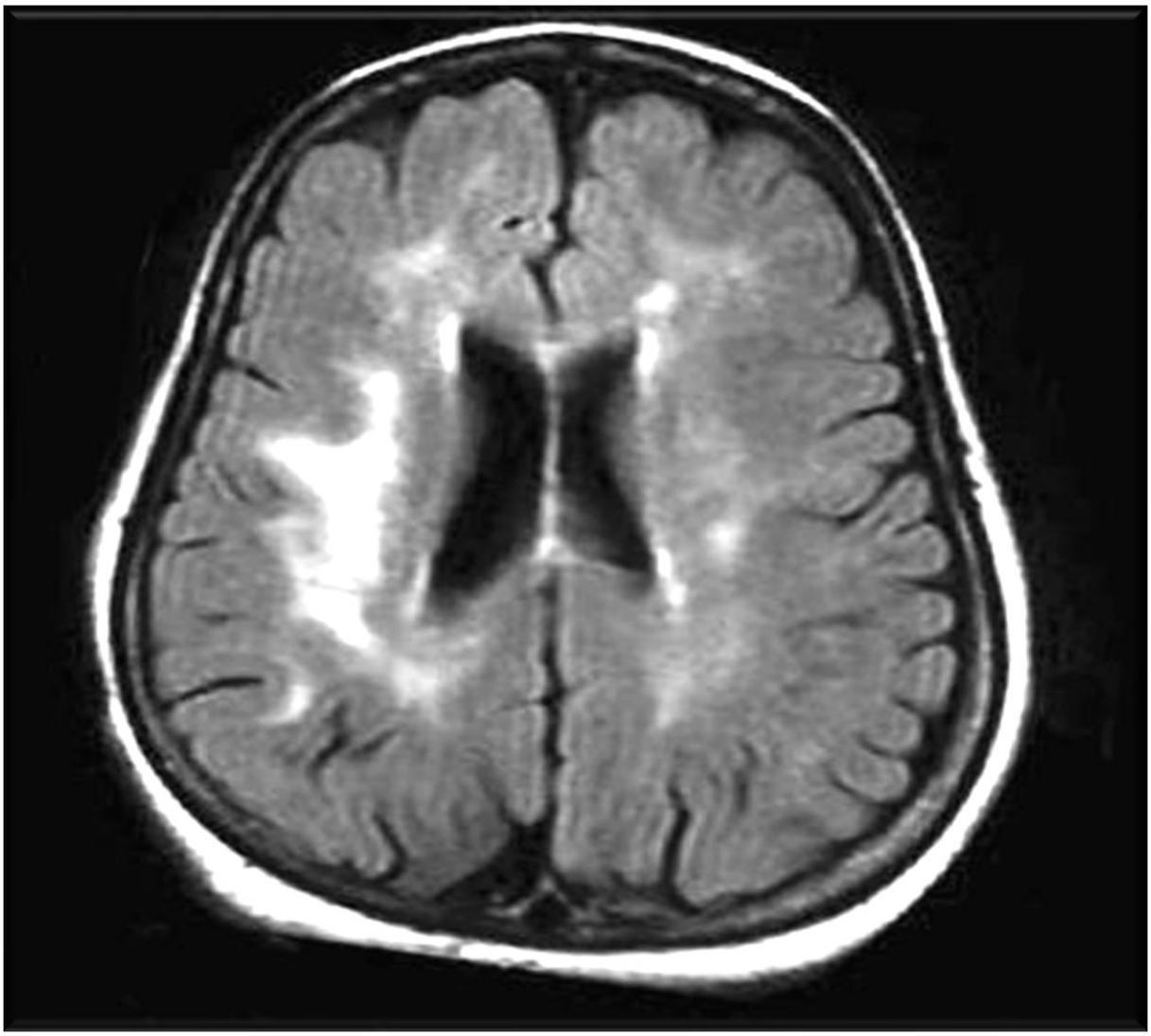

Figure 1. MRI analysis of the ventricular system.

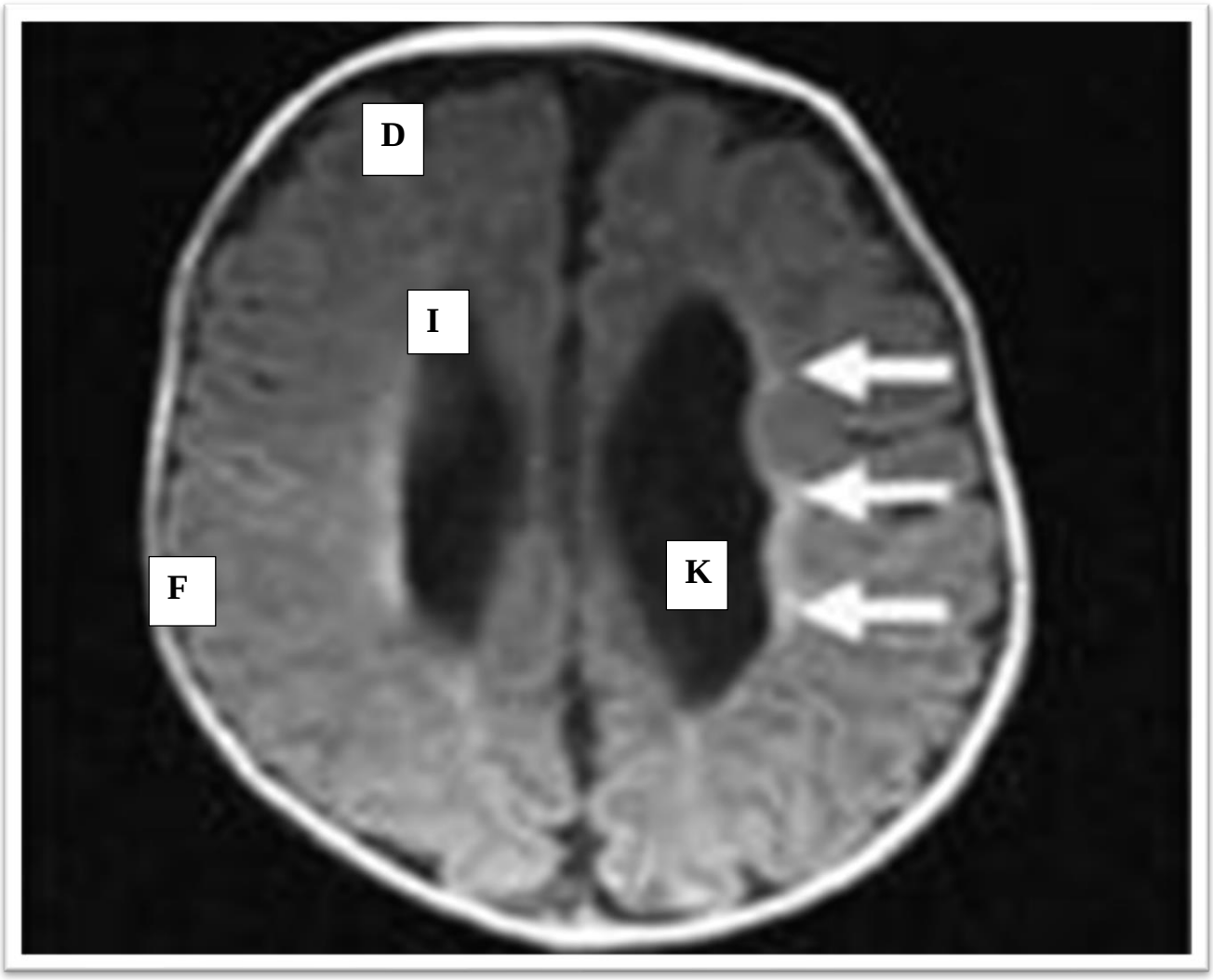


Figure 2. MRI analysis of the ventricular system.

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