OPTIMIZATION OF COGNITIVE DISORDERS IN DEMENTIA USING THE NEUROFEEDBACK THERAPY

[1]Madjidova Ya.N., [2]Azimova N.M., [3]Xusenova N.T., [4]Salikhova S.M.
[1,2,3,4]Tashkent Pediatric Medical Institute, Neurological clinic "Neuromed Service
https://doi.org/10.5281/zenodo.10714723

Abstract. *Dementia is a syndrome caused by an organic lesion of the brain and characterized by disorders in the manistic and other cognitive spheres, including speech, orientation, abstract thinking, and praxis. These violations should be expressed in such a way as to lead to difficulties in everyday life and/or professional activities. Dementia is often accompanied by emotional and affective disorders, but the level of consciousness remains unchanged until the terminal stage of the process. The presence of dementia does not imply the irreversibility of the defect, its progression, global impairment of intellectual functions or the presence of some specific cause of cognitive impairment [1, 2]. The occurrence of dementia is usually unnoticeable (with the exception of post-traumatic, post-anoxic and post-stroke disorders), the course is often progressive, although in some cases it may be stationary and even reversible.*

Keywords: *post-traumatic, post-anoxic and post-stroke disorders, normotensive hydrocephalus, Parkinson's disease, infectious diseases.*

The incidence of dementia increases with age: from 2% in the population under 65 years of age to 20% in people aged 80 years and older [3]. Among people over the age of 65, approximately 9% have mild or moderate dementia, and 5% have severe dementia [1]. It is often quite difficult to distinguish between the initial stages of dementia and forgetfulness in normal aging, an incorrect solution to this problem leads to overdiagnosis of dementia in the elderly. Age-related cognitive impairment is manifested by mild memory impairment and some slowing down of the speed of mental processes. At the same time, there are no significant restrictions in everyday and professional activities due to cognitive impairment. These individuals are often classified as having “benign senile forgetfulness” or “age-related memory impairment” [4]. Follow-up, however, often reveals the presence of progressive dementia. Therefore, this category of patients is shown dynamic follow-up with repeated examination, usually after 6-12 months, which helps to objectify cognitive impairment [4].

The main causes of dementia are Alzheimer's disease and vascular lesions of the brain (vascular dementia), much less often - dysmetabolic disorders, alcoholism, brain tumors, traumatic brain injury, normotensive hydrocephalus, Parkinson's disease, infectious diseases of the central nervous system (CNS), etc. Identification of the cause of dementia is important, since in some situations adequate treatment can lead to the reverse development of disorders or slow down the progression of the pathological process [1, 4, 5]. In addition, predicting the course of the disease based on correct diagnosis allows the patient and his family members to plan their actions.

Thus, the first stage is the diagnosis of dementia, the second is the search for the cause of dementia. A syndromic diagnosis of dementia cannot be made if the patient has a violation of the level of consciousness or the patient's condition does not allow an adequate assessment of his mental status [4]. It is necessary to distinguish between dementia and organic syndromes accompanied by isolated memory impairment or aphasia. Dementia implies an acquired impairment of cognitive functions, therefore, mental retardation (oligophrenia) does not apply to

dementia. The main difference between delirium and dementia is a violation of the level of consciousness, which, however, is not always easy to diagnose with delirium [2]. In addition, with delirium, there is an increase in body temperature, the speech of patients is often dysarthric, and with EEG, a diffuse increase in slow-wave activity is detected. Cognitive impairment in maintaining the level of consciousness in patients with toxic or metabolic disorders is characterized by impaired attention, hallucinations, arousal, motor and speech disorders, and may present certain difficulties in differential diagnosis with delirium [2].

"Pseudodementia" is understood as disorders caused by functional psychiatric disorders (depression, schizophrenia, hysteria), which resemble dementia in their manifestations [1, 6]. The most important among the causes of pseudodementia is depression ("depressive pseudodementia", "cognitive impairment in depression"), which often occurs atypically in the elderly [2, 6]. Depression is characterized by a decrease in mood, appetite, weight loss, sleep disorders (insomnia or hypersomnia), psychomotor agitation or lethargy, fatigue, and the presence of suicidal thoughts. Manifestations of depression in the elderly may include chronic pain syndromes or alcohol abuse. Since most of these symptoms are not specific to depression, a psychiatric consultation may be necessary when assessing the patient's condition. Long-term follow-up of patients with pseudodementia showed that only a small part of them subsequently develop dementia proper [7]. At the same time, there is evidence of a 50% risk of developing dementia over the next few years in elderly patients with depression [8]. It should be noted that pseudodementia often begins suddenly, the symptoms progress rapidly, there may be an indication of the presence of psychiatric pathology in the past, patients make a large number of complaints about their memory, and responses such as "I do not know." Neuropsychological examination in patients with depression reveals disorders mainly associated with attention disorders, the speed of psychomotor processes, and the analysis of details [2]. Mnestic disorders affect both recent and distant events, and there may be significant differences in the results of tasks of almost the same complexity [2, 6]. Unlike patients with dementia, in pseudodementia, memory impairments are mainly due to impaired motivation while maintaining the naming of objects, the ability to count or apraxia tests. However, it should also be taken into account that depression may be one of the first manifestations of developing dementia, in connection with which the term "pseudodementia" raises certain objections [6]. Depression is noted mainly in the early stages of diseases leading to dementia; as the process progresses, accompanied by an aggravation of cognitive functions, depression is much less common [6]. The treatment of dementia is a complex task, including not only drug therapy aimed, if possible, at the underlying disease, but also social and psychological support for patients, patient care [9]. Memory training exercises are ineffective in the advanced stages of dementia, but they can be used in patients with the initial stages of the disease [8,9]. Antidepressants and antipsychotics are used to correct psychotic and behavioral disorders. When prescribing neuroleptics to this category of patients, it should also be remembered about the possibility of having a clinically similar disease of diffuse Levy bodies with Alzheimer's disease, in which the use of neuroleptics, even in small doses, is not indicated [10]. In the presence of depression in patients with dementia, serotonin reuptake inhibitors are currently preferred, since, unlike tricyclic antidepressants, they have less anticholinergic side effects [11]. Central acetylcholinesterase inhibitors (amiridine, rivastigmine, donepecil), peptidergic drugs (cerebrolysin), and nootropic drugs are currently used in Alzheimer's disease. There are some indications of the ability of estrogen replacement therapy, nonsteroidal anti-inflammatory drugs, tocopherol and selegiline to

slow the progression of this disease [9]. In vascular dementia, great importance is attached to the use of drugs that can affect risk factors: antihypertensive agents, disaggregants, anticoagulants according to indications. Patients with dementia, as a rule, are elderly, they often have somatic disorders, which may require joint management of these patients with a cardiologist, pulmonologist, urologist and doctors of other specialties [3]. There are quite a lot of frankly opportunistic statements about the miracle Neurofeedback therapy and stories about "miraculous cures" that are circulating on the Internet, which only harms the prospects for further research on Neurofeedback and the prospects for its use in clinical practice. Meanwhile, researchers in Germany and the Netherlands continue to explore the potential benefits of using Neurofeedback.

The main advantage of Neurofeedback is that it can help patients avoid significant drug load and taking medications that often have side effects. Dr. Norman Doidge, a psychiatrist at the Center for Psychoanalytic Learning and Research at Columbia University and author of the book "The Brain Changing Itself," said he considers Neurofeedback a "powerful brain stabilizer." Whether such results can be achieved in post-stroke patients is a matter of debate. Nevertheless, this technique has been widely recognized by practitioners and an increasing number of patients recognize its effectiveness.

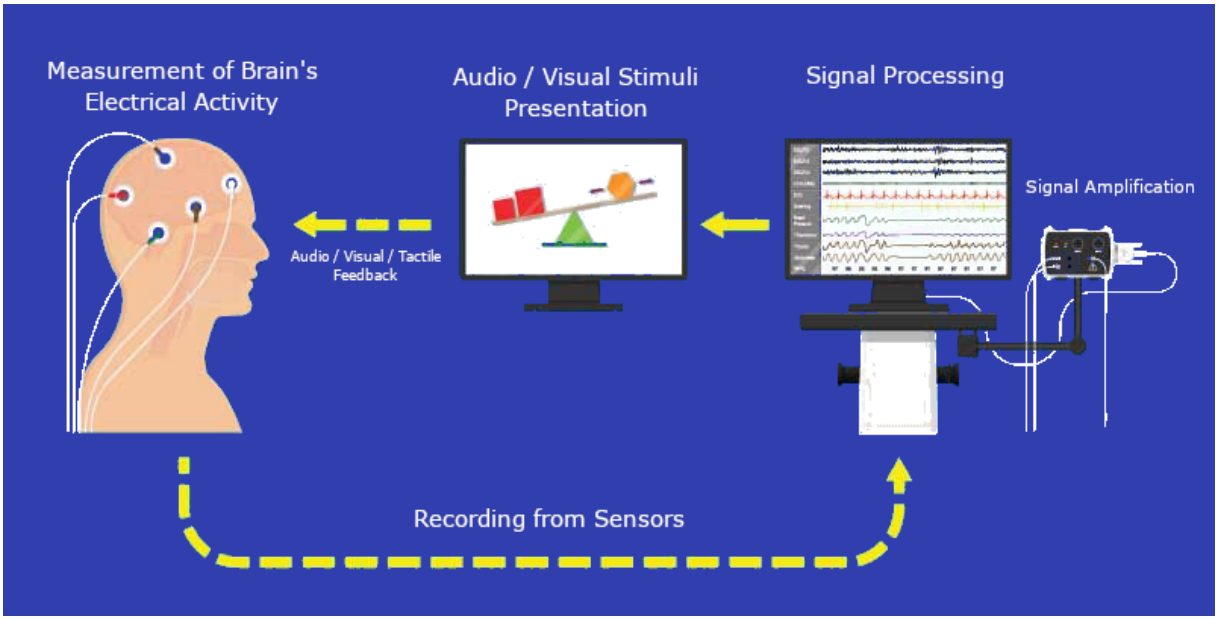


Figure 1. Neurofeedback training process diagram

This format of conducting rehabilitation procedures is most convenient for both the patient and the doctor: one specialist can conduct up to 14-15 such "virtual" patients at the same time.
When automatically setting the threshold of the trained parameter and the actual training, both the dynamics of the trained parameter and histograms of EEG power by ranges, EEG spectra for each time frame of the trained parameter and the training success table are recorded and then stored in the computer memory (Fig. 2).

On the graph of the dynamics of the parameter being trained, vertical lines mark the moments when the adjustment, training and rest cycles are switched. The vertical blue line marks the moment in time for which histograms of EEG power by ranges are displayed on the corresponding graphs. The rehabilitation course lasts for several months. Classes are held daily or every other day, depending on the severity of the disease and the recommendations of the attending physician.

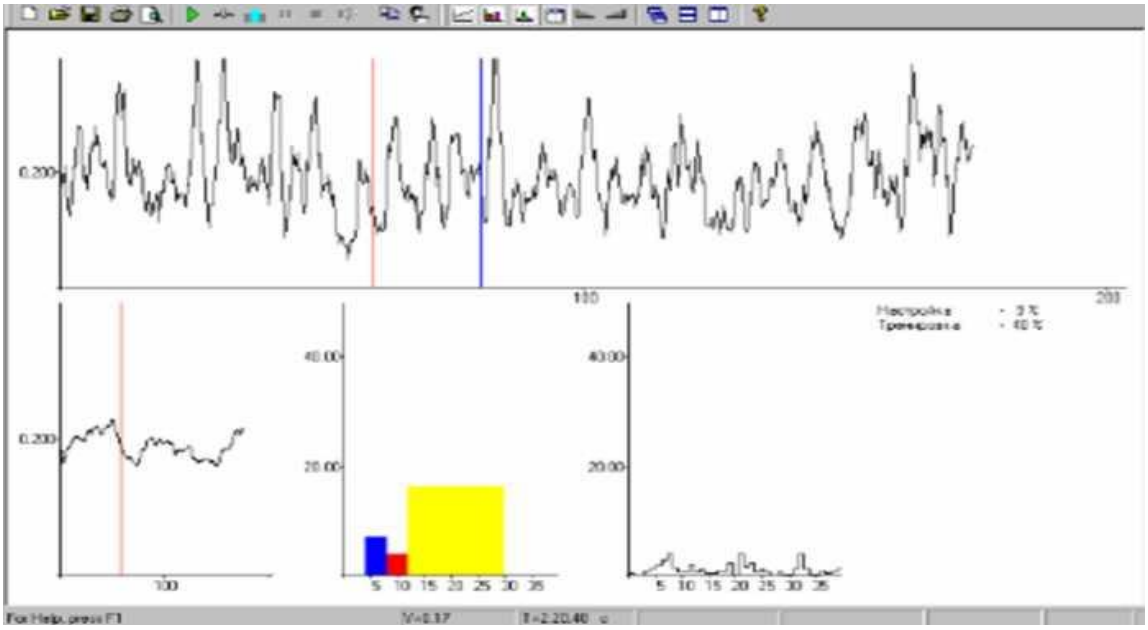


Figure 2. The visual interface of a single training session. The training line moves from left to right during a training session. This line represents a participant's brain activity.

In this work, studies have been conducted on the use of drugs Alcheba, cytoflavin and their combination, as well as a simulator of a psychophysiological state using the MITSAR with Neurofeedback. According to recent literature data, cognitive disorders are corrected by a number of neuroprotective drugs. A special place among them is occupied by the cytoprotector cytoflavin and muscle relaxants of central action (medatilin).

Cytoflavin increases the intensity of aerobic glycolysis, which leads to the activation of glucose utilization and β-oxidation of fatty acids, and also stimulates the synthesis of γ-aminobutyric acid in neurons. Increases the resistance of nerve and glial cell membranes to ischemia, which is expressed in a decrease in the concentration of neurospecific proteins characterizing the level of destruction of the main structural components of the nervous tissue. Improves coronary and cerebral blood flow, activates metabolic processes in the central nervous system, restores impaired consciousness. promotes regression of neurological symptoms and improvement of cognitive functions of the brain. It has a quick awakening effect in post-acute depression of consciousness. When using the drug Cytoflavin in the first 12 hours from the onset of stroke, a favorable course of ischemic and necrotic processes in the affected area (reduction of the lesion), restoration of neurological status and a decrease in the level of disability in the long term are observed.

Alcheba (memantine) is a potential-dependent, medium affinity, non-competitive antagonist of N-methyl-D-aspartate (NMDA) receptors, has a modulating effect on the glutamatergic system. Memantine is a reversible blocker of postsynaptic NMDA glutamate receptors. The use of memantine increases the threshold for generating the excitation potential of the postsynaptic membrane, but does not block the glutamatergic synapse completely. It is known that in Alzheimer's disease, acute and chronic cerebral ischemia and other cerebral diseases with a picture of severe cognitive impairment, the activity of the glutamatergic system increases and more mediator is released into the synoptic cleft and glutamate accumulates in cytotoxic concentrations, triggering the process of excitotoxicity, which leads to the death of neurons. Thus, the use of memantine in cognitive disorders of various etiologies contributes to the normalization of the glutamatergic transmission pattern, which underlies the neuroprotective and positive

symptomatic effect of this drug. Memantine regulates ion transport, blocks calcium channels, normalizes membrane potential, improves the transmission of nerve impulses, improves cognitive processes, memory, concentration, attention and learning ability, increases daily activity, reduces fatigue and symptoms of depression. Memantine also blocks glutamate receptors of the substantia nigra, thereby reducing the excessive stimulating effect of cortical glutamate neurons on the neostreatum, which develops against the background of insufficient dopamine release. It has a neuromodulating effect. In addition to its effect on the central nervous system, memantine affects efferent innervation. It has a greater effect on stiffness (rigidity and bradykinesia), reduces spasticity caused by diseases and brain damage.

The dynamics of treatment was evaluated in 125 patients in the post-stroke recovery period. Depending on the type of therapy performed, the patients were divided into 4 subgroups:

Group 1 consisted of 30 patients receiving two–stage cytoprotective therapy with Cytoflavin for 40 days and Neurofeedback;

The drug cytoflavin 10.0 ml, diluted with 200.0 ml of 5% glucose and injected intravenously for 10 days, then cytoflavin was prescribed in tablets of 1 tab. 3 times a day for 30 days.

Group 2 consisted of 30 patients receiving 2-stage therapy with Alcheba and Neurofeedback (30 days).

The maximum daily dose is 40 mg per day. To reduce the risk of adverse effects, the maintenance dose is achieved by increasing titration to 5 mg per week for the first 3 weeks as follows:

· during the 1st week of therapy (days 1-7) at a dose of 5 mg/day (10 drops) for 7 days
· during the 2nd week (days 8-14) - at a dose of 10 mg / day (20 drops) for 7 days
· during the 3rd week (days 15-21) - 15 mg / day (30 drops) - for 7 days
· Starting from the 4th week - 20 mg / day (40 drops).

The maximum daily dose is 40 mg per day. To reduce the risk of adverse effects, the maintenance dose is achieved by increasing titration to 5 mg per week for the first 3 weeks as follows:

· during the 1st week of therapy (days 1-7) at a dose of 5 mg/day (10 drops) for 7 days
· during the 2nd week (days 8-14) - at a dose of 10 mg / day (20 drops) for 7 days
· during the 3rd week (days 15-21) - 15 mg / day (30 drops) - for 7 days
· Starting from the 4th week - 20 mg / day (40 drops).

Group 3 consisted of 30 patients who received Alcheba, Cytoflavin and Neurofeedback in complex treatment (30 days).

Group 4 included 35 patients with a basic treatment method.

Let's consider the subjective symptoms in the dynamics of treatment for each group separately (Table 1).

As can be seen from the presented data in Table 1, headache was observed with almost the same frequency in all 4 subgroups. After treatment, headache regression was observed, so in group 1 by 26.6%, in group 2 by 26.7%, in group 3 by 33.3% and in group 4 by 5.7%. After treatment, the regression of dizziness was noted in group 1 by 13.4%, in group 2 by 20%, in group 3 by 16.6% and in group 4 by 8.3%. The incidence of impaired memory after treatment decreased in group 1 by 63.3%, in group 2 by 66.7%, in group 3 by 73.3% and in group 4 by 11.4%. The same trend is

observed in the presence of complaints of tinnitus, increased fatigue, decreased performance and attention, and sleep disturbances.

Table 1

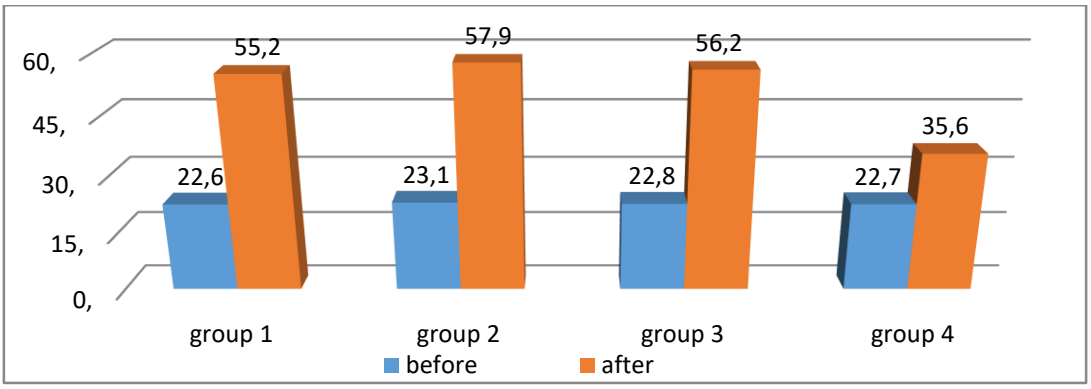
Subjective symptoms of post-stroke patients depending on the type of therapy

		group 1, n=30	group 2, n=30	group 3, n=30	group 4, n=35
Headache	Before	19 (63,3%)	18 (60%)	18 (60%)	21(60%)
	After	11 (36,7%)	10 (33,3%)*	8 (26,7%)*	19(54,3%)
Vertigo	Before	11 (36,7%)	12 (40%)	10 (33,3%)	14 (40,0%)
	After	7 (23,3%)	6 (20%)**	5 (16,7%)**	11 (31.7%)
Impaired memory	Before	30 (100%)	30 (100%)	30 (100%)	35 (100%)
	After	11(36,7)	10 (33,3%)**	8 (26,7%)**	31 (88,6%)
Tinnitus	Before	20 (66,7%)	20 (66,7%)	19 (63,3%)	23 (65,7%)
	After	11 (36,7%)*	10 (33,3%)*	9 (30%)**	19 (54,3%)
Increased fatigue	Before	29 (96,7%)	29 (96,7%)	28 (93,3%)	34 (97,1%)
	After	14 (46,7%)	13 (43,3%)*	12 (40%)*	24 (68,6%)
Reduced performance	Before	30 (100%)	29 (96,7%)	30 (100%)	35(100%)
	After	11 (36,7%)*	10 (33,3%)*	6 (20%)*	25 (71,4%)
Reducing attention	Before	29 (96,7%)	28 (93,3%)	28 (93,3%)	33 (94,3%)
	After	11 (36.7%)	10 (33,3%)*	9 (30%)*	21 (60%)
Sleep Disturbance	Before	26 (86,7%)	19 (63,3%)	25 (83,3%)	30 (85,7%)
	After	14 (46,7%)	13 (43,3%)*	12 (40%)*	24 (68,9%)

Note: * - reliability of data between pre-treatment and post-treatment parameters (P<0.05-0.01)

Thus, according to the data obtained, in patients in the post-stroke period, the inclusion of alchebo in the treatment complex, as well as its combination with cytoflavin and Neurofeedback, is most effective. It should also be noted that there is a fairly pronounced regression of subjective symptoms when the drug cytoflavin is included in the treatment in combination with Neurofeedback. When studying the data of cognitive function in patients in the early post-stroke recovery period after treatment, a positive dynamics of leveling cognitive function disorders was established, depending on the methods of therapy. Note that the maximum possible score on the Barthel scale is 100 points. The average value (in points) of the patients' assessment on this scale at the beginning of treatment was 22,60±1,5, 23,1±1,2, 22,8±1,4 and 22.7±1.4 points, respectively, in groups. 3 months after the treatment measures, the Barthel scale indicators changed dramatically

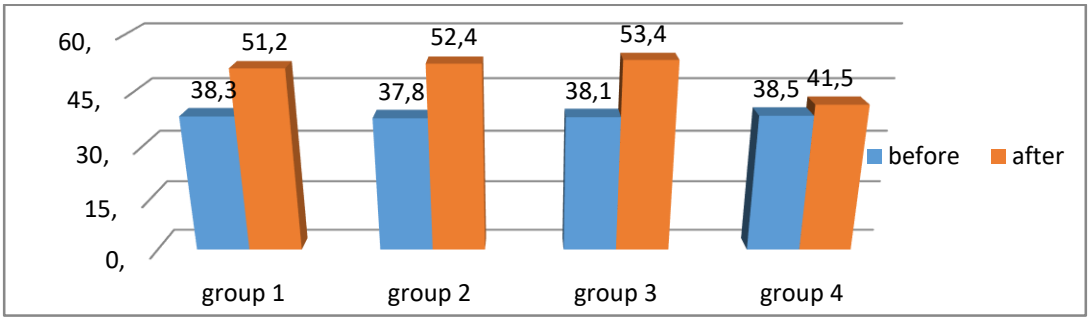
in groups 1, 2 and 3, however, in group 4 after the treatment, the indicators tended to positive dynamics. The difference in scores at the beginning of treatment and after it averaged 32.6, 34.8, 33.4 and 12.9 points (Fig. 3).



Note: * - reliability of data before and after treatment (P<0.05);

Fig. 3. Dynamics of indicators on the Barthel scale during treatment

A comparison of the severity of focal neurological deficit according to SNS showed that patients have a high degree of focal neurological deficit (Fig. 4).



Note: * - reliability of data before and after treatment (P<0.05);

Fig. 4. Dynamics of indicators on the SNSS scale during treatment

As can be seen from the diagram, in patients of the main group and the control group, there is a tendency to level neurological deficits. However, it was more pronounced in groups 1, 2 and 3. The difference in scores before and after treatment was 12.9 points in group 1, 14.6 in group 2, and 15.3 in group 3, which is higher than 4.3 in relation to group 4 (3.0 points; P<0.05).

It should be noted that, despite the absence of a statistically significant advantage in the main group on the FAB scale, reflecting the state of the functions of the frontal-subcortical brain, after a course of restorative training, there was a more pronounced dynamics in the performance of tasks characterizing both visual-spatial processes and the functions of the mediobasal temporal divisions, as well as the interest of subcortical-frontal connections the brain (Table 2).

Table 2

Dynamics of data on the FAB and MoCA scale

Groups	FAB		MoCA	
	Before	After	Before	After
group 1	14,2±0,2	16,9±0,1*	20,3±0,1	24,2±0,2*
group 2	13,8±0,3	15,8±0,2*	19,9±0,2	24,5±0,1*
group 3	13,9±0,2	17,0±0,3*	20,2±0,3	25,1±0,3*

group 4	14,3±0,2	14,4±0,2	20,1±0,1	22,2±0,1

Note: * - reliability of data before and after treatment (P<0.05);

When conducting the MoCA test in groups 1, 2, 3, compared with group 4, better results were achieved in the subscales "attention" and "memory", which are not directly related to the stimulated area of the brain.

Thus, cognitive impairment was observed before treatment in the form of pre—dementia disorders in the main group in 64.4%, in the comparison group in 63.8%, mild dementia in 28.9% and 28.8% of cases, respectively (Table 3).

Table 3

Dynamics of detection of pre-dementia and dementia disorders in the course of treatment

Groups	Pre-dementia disorders				Dementia disorders			
	Before		After		Before		After	
	Abs.	%	Abs.	%	Abs.	%	Abs.	%
group 1 (n=30)	20	66,7	10	33,3*	8	26,7	4	13,3*
group 2 (n=30)	21	70,0	11	36,7*	9	30,0	5	16,7*
group 3 (n=30)	19	63,3	7	23,3*	10	33,3	4	13,3*
group 4 (n=35)	24	68,6	15	42,9	10	28,6	7	20,0

Note: * - reliability of data before and after treatment (P<0.05)

After the treatment measures in patients of groups 1,2. 3, the incidence of pre–dementia disorders decreased by almost 2 times, in group 4 these indicators were 68.6% (24 patients) versus 42.9% (15 patients). The incidence of pre-dementia disorders in groups 1, 2, and 3 decreased by 31.4%, 33.3%, and 40%, respectively, and in group 4 by 26% (P<0.05). Thus, the results of repeated testing after the course of treatment showed that in groups 1, 2, 3, there was a statistically significant improvement in cognitive functions in the dynamics of treatment ($p < 0.05$), in the fourth group there was a tendency to improve in the cognitive sphere.

The effectiveness of treatment was assessed on the CGI scale (Clinical Global Impression). The Clinical Global Impression Scale was created in 1976 at the National Institute of Mental Health in the United States for collaborative programs on the study of schizophrenia. It consists of 3 subscales reflecting the assessment of the severity of the condition, the overall degree of its improvement according to a 7-point system, and a subscale of the effectiveness index, which is calculated based on the combination of one of the four degrees of therapeutic effect (noticeable, moderate, minimal, unchanged) and the severity of the side effect of the drug (absent, insignificant, significant, leveling therapeutic effect) [12].

By the 30th day of treatment, the most significant results were obtained in group 3, so marked improvement was observed in 63.3% of cases, moderate and minimal improvement was registered in 26.7% and 10.0%, respectively. Almost identical results were obtained in groups 1 and 2. In group 4, insignificant treatment results were noted, so the lack of improvement and minimal deterioration in this group amounted to 11.6% and 4.7%, respectively (Table 4).

Table 4

The effectiveness of treatment of patients with MCI on the background of CCI, who were on different treatment methods by the 14th day of treatment

Groups		Marked improvement	Moderate improvement	Minimal improvement	No improvement	Minimal deterioration
1	abs	21	8	1	-	-
	%	70	26,7	3,3	-	-
2	abs	26	3	1	-	-
	%	86,7	10	3,3	-	-
3	abs	23	5	2	-	-
	%	76,7	16,7	6,7	-	-
4	abs	20	8	5	1	1
	%	57,1	22,9	14,03	2,9	2,9

Particularly pronounced efficacy of treatment was noted in post-stroke patients of group 3 who took concomitant administration of the drugs Alcheba and cytoflavin, as well as Neurofeedback.

Thus, it can be concluded that the data obtained on the differentiation of the therapeutic response depending on the clinical features of cognitive deficits contribute to optimizing therapy in order to suspend or slow its progression and maintain the quality of life of both post-stroke patients and their families.

The economic efficiency of the proposed treatment method for each patient averaged 324,000 sums, due to a decrease in the number of medications taken at the outpatient stage of treatment, whereas with the traditional method of therapy, the costs average 525,000 sum, which is 1.6 times higher (72,000 sum).

Conclusions. 1. The obtained data on the comparative effectiveness of the studied types of therapy contribute to solving complex psychopharmaceutical problems that arise when it is necessary to choose certain methods of drug action on the manifestations of dementia syndrome.

2. The developed indications for the prescription of drug therapy allow for a targeted effect on cognitive deficits in the early post-stroke period in accordance with the established spectrum of action of the studied drugs.

3. A patient in the early post-stroke period with pre-dementia disorders should include Alchebo (30 days) and Neurofeedback (30 days) in the treatment package.

4. Patients with dementia in the early post-stroke period in the treatment complex should be prescribed the combined use of cytoflavin and Alchebo drugs, as well as with the inclusion of

Neurofeedback. The drug cytoflavin 10.0 ml, diluted with 200.0 ml of 5% glucose and injected intravenously for 10 days, then cytoflavin was prescribed in tablets of 1 tab. 3 times a day for 30 days. Alchebo was prescribed for 30 days:

The maximum daily dose is 40 mg per day:

· during the 1st week of therapy (days 1-7) at a dose of 5 mg / day (10 drops) for 7 days
· during the 2nd week (days 8-14) - at a dose of 10 mg / day (20 drops) for 7 days
· during the 3rd week (days 15-21) - 15 mg / day (30 drops) - for 7 days
· Starting from the 4th week - 20 mg / day (40 drops).

REFERENCES

1. Kispaeva T. T., Ivanova G. E., Volchenkova O. V., Samsygina O. M. Principles and methods of cognitive rehabilitation of patients in the acute period of cerebral stroke. Physical therapy and sports medicine. 2009; 7 (67): 48–57.
2. Kovalchuk V. V., Skoromets A. A., Kovalchuk I. V., Stoyanova E. G., Vysotskaya M. L., Melikhova E. V., etc. The use of the drug Acatinol Memantine in patients with vascular cognitive disorders. Journal of Neurology and Psychiatry named after C. C. Korsakov. 2010; 12:37–40.
3. Levin O. S. Diagnosis and treatment of dementia in clinical practice: 4th ed. Moscow: MEDpress-inform; 2014. 256 p.
4. Majidova E.N., Rasulova H.A. Neuroprotection in angioeducology//Tashkent.– 2009. – 107s.
5. Prokopenko S. V., Mozheyko E. Yu., Levin O. S., Koryagina T. D., Chernykh T. V., Berezovskaya M. A. Cognitive impairment and their correction in the acute period of ischemic stroke. Journal of Neurology and Psychiatry named after S. S. Korsakov. 2012; 112 (8): 35-9.
6. Prokopenko S. V., Mozheyko E. Yu., Shvetsova I. N. A method for restoring cognitive functions in cerebrovascular pathology: patent of the Russian Federation No. 2586312. The patent holder of KrasSMU, declared on 12/20/2011, published on 02/20/2014. Byul. No. 16.
7. Tsukurova L. A., Esipenko N. V. Improving the target indicators of morbidity, mortality and mortality from strokes in the Krasnodar Territory // Russian Medical Journal. 2012. No. 10 . pp. 534-536.
8. A Alonso, T H Mosley, Jr, R F Gottesmanet all. Risk of dementia hospitalisation associated with cardiovascular risk factors in midlife and older age: the Atherosclerosis Risk in Communities (ARIC) study. J NeurolNeurosurg Psychiatry 2009;80:1194–1201
9. Forette F., Rockwood K. Therapeutic intervention in dementia. /In: Diagnosis and management of dementia. A manual for memory disorders team. Ed. by G.Wilcock, R.S.Bucks, K.Rockwood. - Oxford, N.Y.k: Oxford University Press, 2009 -P.294-310
10. Guk R. Yu., Jafarova O. A., Sklyar M. M., Chernikova L. A., Tarasevich A. F., Shtark M. B. Neurofeedback and network rehabilitation. // Proceedings of 17th ESPRM European Congress of Physical and Rehabilitation Medicine. Venice, Italy, May 23—27, 2010. P. 28—30.
11. Jafarova O. A., Tarasov E. A., Shtark M. B. Development of the system for continuous medical rehabilitation for patients with post-stroke and spinal cord injury motor disorders // Proceedings 9th Intl Conf. on Disability, Virtual Reality and Assoc. Technologies, P M Sharkey, E Klinger (Eds). Laval, France, Sept.10—12, 2012. P. 385—392.

12. Jaillard A., Naegele B., Trabucco-Miguel S., LeBas J., Hommel M. Hidden functioning in subacute stroke. Stroke.
13. Kolevzon A., Ventola P. Development of an adapted Clinical Global Impression scale for use in Angelman syndrome. // Journal of neurodevelopmental disorders DOI:10.1186/s11689-020-09349-8., 2021