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ASSESSING THE SEVERITY OF NEUROLOGICAL DISORDERS AND COMPARING THEM WITH THE DEGREE OF STATODYNAMIC FUNCTIONAL DISORDERS

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Abstract:

the basic pathogenetic links of the occurrence of postural disorders are for the most part quantifiable in clinical settings, which allows for a systematic analysis, with the help of which it is possible to switch from non-specific methods of treating patients, often still based on empirical observations, to specific ones, based on targeted therapeutic effects on key mechanisms of development of various diseases, manifested by statodynamic disorders. In this regard, further research is needed to identify the key pathophysiological mechanisms responsible for the development of various clinical forms of imbalance, which will effectively apply fundamental knowledge about the basic biological mechanisms of locomotion in clinical practice.

Keywords: Cavinton, mathematical modeling of statodynamic disorders.

Introduction

Therapy of imbalance is an extremely difficult task due to the large number of pathogenically heterogeneous diseases manifested by loss of statodynamic control. Currently, the following pharmacological agents are used: vestibular suppressants (anticholinergic, antihistamines and benzodiazepines), glucocorticosteroids (vestibular neuritis, Meniere's disease), antidepressants (persistent postural-perceptual dizziness, vestibular migraine), beta-blockers and anticonvulsants (vestibular migraine), diuretic acetazolamide (Meniere's disease, episodic ataxia 2 type), 4aminopyridine (cerebrospinal ataxia), muscle relaxant baclofen (nystagmus syndrome, beating down), piribedil (postural instability), etc.

One of the most important areas of pathogenetic therapy for patients with statodynamic disorders is the stimulation of neuroplasticity, which allows modulating the functioning of neural networks responsible for maintaining body balance, thereby creating the basis for effective sanogenesis and compensation of impaired functions. This approach is particularly justified in the treatment of

patients with cerebrovascular pathology (dyscirculatory encephalopathy and the consequences of acute cerebral circulatory disorders), neurodegenerative diseases, persistent postural-perceptual vertigo, vestibular neuronitis and a number of other diseases.

Among the non-drug methods of activating neuroplasticity, it is of great importance to perform vestibular gymnastics exercises that compensate for statodynamic disorders based on three principles: adaptation (remodeling of neuronal connections), substitution (strengthening the role of "healthy" sensory signals in maintaining balance control) and habituation (increasing the threshold for sensory stimuli). The analysis of the results of these exercises demonstrates their high effectiveness in patients with chronic dizziness. This is especially true for the ability to self-serve and improve the quality of life. At the same time, an individual program of vestibular gymnastics (rehabilitation) based on a comprehensive examination is the most effective.

Of the pharmacological agents capable of inducing neuroplastic processes in the brain, vinpocetine (Cavinton) should be isolated. The active substance of Cavinton is the ethyl ether of apovincamic acid. The clinical effect of the drug is based on the inhibition of phosphodiesterase (Ca/calmodulin-dependent type 1) and potential-dependent Na+ channels, which allows it to directly act on glutamate receptors, inhibit lipid peroxidation, cause vasodilation, inhibit platelet aggregation and increase the deformability of erythrocytes Vinpocetine has a normalizing effect on the arteries of the brain both with increased, and decreased tone, restoring the ability to autoregulate cerebral circulation and preventing the development of vasoconstrictor reactions. In addition, Cavinton prevents neuronal death in the hippocampus region, stimulates the noradrenergic system of the ascending reticular formation [40] and has a modulating effect on neuroplasticity (increases the growth of dendritic spines) [41].

In an open clinical trial, we evaluated the effectiveness of Cavintone comfort (a new dispersible form) in combination with vestibular gymnastics exercises in the treatment of chronic vertigo in patients with dyscirculatory encephalopathy [42]. In the group of patients receiving Cavinton forte as part of complex therapy, after 3 months there were statistically significant positive changes in all the studied parameters – the duration and severity of dizziness (VASH), increased motor activity of patients during the day (the scale for assessing the effect of dizziness on daily activity – Dizziness Handicap Inventory [DHI]), increased adherence to treatment (Drug Attitude Inventory [DAI]). It is important that the level of brain neurotrophic factor (BDNF) in the blood plasma of patients in this group increased almost 3 times compared to the baseline value. This fact indicates that the basis of the therapeutic effectiveness of the drug is the modulation of neuroplasticity of the brain.

In 2019, we studied the effectiveness of long-term use of Cavintone Comfort in the treatment of dizziness in patients with dyscirculatory encephalopathy (EDELWEISS study) [43]. The most significant predictors of the development of statodynamic disorders in this category of patients were the age over 70 years, the total score of cognitive functions on the Montreal scale <25 (Montreal Cognitive Assessment, MoSA) and low blood BDNF (<10pg/ml). These results indicate that age alone is not the only factor determining the development of postural instability and dizziness in patients with dyscirculatory encephalopathy, this is also noted by other researchers [44]. After 2 months of therapy in a group of patients receiving Cavinton® For example, we observed a significant decrease in the intensity of dizziness (according to YOUR-G) and its effect on daily activity (according to the DHI scale) compared with the initial parameters, and after 3 months – compared with the control group. The tendency to an even more pronounced subjective improvement in the condition of patients persisted for 5 months of therapy (maximum clinical effect), and then inverted. Nevertheless, even 2 months after the completion of the 6-month course of taking vinpocetine, patients in the main group had a significantly less pronounced feeling of dizziness and a decrease in its effect on daily activity compared with the baseline level and corresponding indicators in the control group. It is important to note that the dynamics of

improvement in patients with dyscirculatory encephalopathy according to the DHI scale significantly correlated (Pearson correlation coefficient >0.5) with an increase in BDNF expression in blood plasma throughout the entire observation, which indicates the presence of a clear link between subjective improvement and activation of neuroplastic processes in the brain. In addition to the subjective change in the condition of patients, according to videonystagmography data, after 3 months of therapy, an objective improvement in the indicators of the smooth tracking test and the saccade test was revealed compared with the initial values and corresponding results in patients of the control group. The same pattern was noted when evaluating both tests and after 6 months of the study, and for the saccade test – even 2 months after the withdrawal of vinpocetine (at the end of the 8th month of follow-up).

Thus, the clinical manifestations of the functional inferiority of the CDC are diverse, which is due to the complexity of its organization. Nevertheless, the basic pathogenetic links of the occurrence of postural disorders are for the most part quantifiable in clinical settings, which allows for a systematic analysis, with the help of which a transition from non-specific (symptomatic) methods of treating patients (for example, vestibular suppression), often still based on empirical observations, to specific ones, based on which have a targeted therapeutic effect on the key mechanisms of the development of various diseases manifested by statodynamic disorders. In this regard, further research is needed to identify the key pathophysiological mechanisms responsible for the development of various clinical forms of balance disorders (including mathematical modeling of statodynamic disorders in humans using experimental data), which will effectively apply fundamental knowledge about the basic biological mechanisms of locomotion in clinical practice.

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