Pathogenetic and Clinical Features of the Recovery Period of Ischemic Stroke

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Abstrak. The data of a number of authors indicate that the first six months after the onset of stroke are fundamental in relation to the formation of the final neurological deficit, which determines the level of functional and everyday capabilities of the patient. Despite the fact that by the 3rd-4th week of stroke, the formation of a heart attack site is completed, a number of processes started at the very beginning of the disease, primarily the mechanisms of programmed cell death - apoptosis, permeability of the blood-brain barrier, and microcirculation disorders remain important. In the first 2 months after the onset of stroke, reparative and regenerative processes occur associated with collateral blood circulation in the area of injury, regression of edema, and absorption of necrotic tissues.

Relevance. The data of a number of authors indicate that the first six months after the onset of stroke are fundamental in relation to the formation of the final neurological deficit, which determines the level of functional and everyday capabilities of the patient. Despite the fact that by the 3rd-4th week of stroke, the formation of a heart attack site is completed, a number of processes started at the very beginning of the disease, primarily the mechanisms of programmed cell death - apoptosis, permeability of the blood-brain barrier, and microcirculation disorders remain important. In the first 2 months after the onset of stroke, reparative and regenerative processes occur associated with collateral blood circulation in the area of injury, regression of edema, and absorption of necrotic tissues. The described data from molecular biology and pathomorphology indicate the expediency of limiting this time interval during an ischemic stroke. Several classifications of clinical periods of ischemic stroke are presented, each of which highlights the early recovery period. This time interval is characterized by maximum recovery of lost functionality. This period ends in two months, but the end of the acute and, consequently, the beginning of the early recovery period depends on the severity of the stroke. With non-severe cerebral injury, the acute period ends at the end of the second week, with moderate - at the end of the third, and with severe stroke at the end of the fourth week. The time interval from two months to a year in ischemic stroke is called the recovery period. According to the authors, this period of time is characterized by the formation of a permanent neurological deficit due to the continued increase in functional capabilities, however, the rate and volume of this increase are less pronounced than in the early recovery period. Pathophysiological and clinical studies indicate the expediency of limiting the early recovery period to three weeks to six months. During these periods, compensation for impaired functions occurs, the main mechanism of which is functional restructuring, the involvement of new brain structures in the damaged system and the formation of new associative connections. According to modern scientific concepts, the pathogenesis of stroke and the restoration of impaired functions are considered taking into account the processes of neuroplasticity. Neuroplasticity is characterized by the ability to change its structural and functional organization under the influence of external and internal factors.

Reparative-regenerative processes in the damaged area and compensation of impaired functions occur due to the reorganization of the central nervous system. A component of neuroplasticity is synaptic plasticity, which is the property of synapses to respond to physiological and pathological influences by changing the effectiveness of transynaptic information transmission. Synaptic plasticity of the brain consists in selective action on ion channels, receptors, and various levels of the intracellular regulatory system (calcium, phosphoinositide, adenosine, and guanosine monophosphate), contributing to an increase in the adaptive capabilities of interneuronal transduction. As a result of changes in the threshold of excitability of potential-dependent membrane channels, potentiation or suppression occurs, regulating

the efficiency of synaptic transmission. In recent decades, the object of research has mainly been post-stroke depression, while other affective disorders such as anxiety, fatigue and apathy have been covered only indirectly, and to date, questions of pathogenesis remain open and, consequently, little-known methods of their treatment. The importance of this problem is due to the high incidence of this complication. An analysis of one long-term study of stroke survivors showed that the incidence of depression in stroke patients is 34%, compared with 13% in the general population. However, it is worth noting that there is a very wide variation in the incidence of depression after a stroke. According to various authors, the incidence of post-stroke depression ranges from 11 to 72%. Such a significant difference in the incidence of post-stroke depression is related to the diagnosis and assessment period for stroke. Along with functional changes, structural transformations also occur in the synapse. Microbiological studies indicate changes in the number of synapses, the length and configuration of their active zones, and changes in the number of spines on the processes of a neuron as a result of axonal or dendritic springing. Studies have also shown that changes in astroglia, namely structural changes in astrocytes, and an increase in the number of contacts between synapses and astrocytes, are of no small importance in the realization of neuronal plasticity. It is known that neuroplasticity stimulates the repeated performance of certain movements, which helps to consolidate the stereotype of the function and reorganization of the cerebral cortex. The phenomenon of long-term potentiation, which consists in prolonged neural activation of certain structures after tetanization of its afferent input, is of great importance for the realization of neuroplasticity. Due to this phenomenon, learning and memory processes are carried out. Plasticity perpetuates the changes that occur in the nervous system during its interaction with the environment, and consolidates the emerging new connections, systems, and intersystem relationships.

In animal experiments, there is evidence that the effectiveness of restorative treatment depends on the environment. Studies by K. Puurunen and J. Sivenius (2002) have shown the beneficial effect of a decent environment on improving the household adaptation of rats due to increased plasticity of neuronal synapses and an increase in their number. Similar conclusions were drawn based on the results of other studies that showed the dependence of the representation of motor cells of the affected limb of monkeys on the intensity of training, that rehabilitation of stroke patients is based on the reserve capabilities of the brain and the reorganization of its structures in the conditions of the pathological process. This reorganization contributes to the patient's motor retraining, which, in turn, leads either to recovery or to compensation for impaired functions. According to a number of authors, the localization of various centers in the brain is not permanent, and cortical connections are able to be remodeled, involving even currently inactive structures in the process. The patient's compensatory capabilities depend on the lesion of functionally significant areas, the depth and lateralization of the lesion. A good regression of motor symptoms was described with foci in the cortical zone of the motor cortex of the major hemispheres, while a more persistent and pronounced motor defect was with deep localization of the focus (Buteflsch SM, Netz J., Webling M. et al., 2003). A clinical and computer comparison in stroke patients revealed that the severity of motor disorders is largely determined by the location of the lesion in relation to functionally significant areas (pyramidal tract, inner capsule), and to a lesser extent by the size of the lesion. It is advisable to distinguish three levels in the process of rehabilitation treatment. The first level is characterized by the true restoration of impaired functions to their original state. Compensation, which is the second level, consists in the functional transformation of unaffected brain systems. Readaptation is the third level of recovery underlying social rehabilitation aimed at adapting to the residual anatomical and functional deficit. There is an undeniable need to strive for true recovery of patients' functions after a stroke, in which the altered functions are fully restored or as close as possible to their original level. However, such a recovery is possible when the pathological focus consists mainly of inactivated elements due to edema, hypoxia, changes in the conduction of nerve impulses, and dialysis, that is, there is no complete death of neurons.

The effectiveness and duration of the therapeutic effect were determined, and technologies (physiotherapy, physical therapy, reflexology, and botulinum therapy) for medical rehabilitation of post-stroke arm and leg spasticity were developed, taking into account the period of the disease. It has been established that in any period of cerebral stroke, the greatest decrease in muscle hypertension (improvement of passive limb function) is observed during botulinum therapy. However, in the acute and early recovery period of cerebral stroke, medical rehabilitation of arm muscle spasticity using botulinum therapy significantly worsens the active function of the limb. It has been shown that in the late recovery and residual period of cerebral stroke, medical rehabilitation of arm muscle spasticity without the use of botulinum therapy does not provide maximum therapeutic effect both in terms of effectiveness and duration of positive clinical effect. It was revealed that in the acute and early recovery period of cerebral stroke, the use of complex technologies for medical rehabilitation of uncomplicated leg spasticity in combination with botulinum therapy or botulinum therapy in the form of monotherapy worsens the active

function of the limb, which is clinically characterized by an increase in the severity of paresis and difficulty in independent movement of the patient, and in the late recovery and residual period of the disease does not significantly affect the active a function. Medical rehabilitation technologies have been developed and the effectiveness and duration of the clinical effect in post-stroke mixed spastic muscle hypertension of the arm and leg with the addition of plastic rigidity, as well as in complicated variants of the course of muscle spasticity, have been evaluated. It has been established that in order to achieve the greatest therapeutic effect in patients with mixed spastic muscular hypertension, regardless of the period of the disease, it is necessary to use complex technologies of medical rehabilitation in the form of a combination of physical therapy, physiotherapy and mandatory botulinum therapy, and in complicated cases of muscular hypertension, additional inclusion of reflexotherapy procedures. The degree of restoration of the active and passive function of the paretic limbs with a decrease in muscle hypertension was assessed. It has been shown that in order to maximize the restoration of active limb function, rehabilitation measures should be carried out as early as possible from the onset of the disease, and improvement of the passive function of paretic limbs does not depend on the timing of medical rehabilitation. It was found that the results of the performed medical rehabilitation do not differ in the effectiveness and duration of the therapeutic effect, depending on the type of cerebral stroke. It has been shown that in any period of cerebral stroke, after comprehensive rehabilitation measures, a gradual re-increase in muscle hypertension occurs.

Conclusion. With more severe cerebral injuries with necrosis of the nervous tissue, the restoration of functions and an increase in the level of household and social activity are more associated with adaptive changes, when the patient learns to compensate for neurological disorders, and the environment undergoes changes leading to maximum independence and independence. In some individuals, despite the continuing neurological deficit, due to readaptation, there may be a restoration of the ability to perform actions necessary for daily life (to eat, dress, wash, use the toilet). Rehabilitation in such patients is aimed at forming a cognitive behavior strategy in the brain to maximize compensation for existing neurological deficits.

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