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TRANSCRANIAL MICROPOLARIZATION IN REHABILITATION OF PATIENTS WITH STROKE

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Annotation
Stroke remains one of the major health problems worldwide, as it is the second leading cause of death and disability. The problem of stroke rehabilitation is not only medical, but also of great social importance, since stroke in many countries is the third most common cause of death and the first most common cause of disability in middle-aged and elderly people. Uzbekistan, the incidence of cerebral stroke ranges from 0.9 to 1.5 per 1000 population (Rakhimdzhanov A.R., 1997; Kilichev I.A., 1998; Gafurov B.G., 2004). Residual disability due to MI in Uzbekistan is 3.2 persons with disabilities per 10,000 population. At the same time, according to many authors, in the last decade, the Central Exhibition Hall has become significantly “younger”, and intellectual workers in their prime are increasingly suffering. The likelihood of developing motor and cognitive impairments in stroke patients is high, which confirms the relevance of the development of rehabilitation measures at the present time.

Keywords: cerebral, hyperreaction, social outcomes, pyramidal syndrome, cranial nerves, middle level, cerebellar ataxia, hyperkinesis.

Introduction

Acute cerebral circulatory disorders are the second most important cause of mortality in the structure of general mortality of the population, second only to cardiovascular pathology. In this regard, interest in the issues of cerebral blood flow disorders does not weaken all over the world, more and more new aspects of this problem are being considered [5,12].

About 85-88% of cerebral strokes are ischemic in nature. Ischemic stroke (IS) is one of the main causes of death in developed countries and ranks 1st as a cause of permanent disability; 85% of patients who have had a stroke remain disabled, and only 10-15% return to their previous work activities. According to the literature, even

after a thorough examination, the cause of IS cannot be established in 40% of cases [6,9]. Hyperreaction of the cardiovascular system during prolonged exposure to mental or biological stress plays one of the important roles in the occurrence of cardiovascular pathology [11].

Cardiovascular pathology and stroke are in first and second place, respectively, as the causes of mortality in the structure of general mortality of the population. In this regard, interest in the issues of cerebral blood flow disorders does not weaken all over the world, more and more new aspects of this problem are being considered [6]. Stroke is the most severe form of cerebral vascular pathology. In European countries, the mortality rate for stroke varies from 63.5 (Switzerland) to 273.4 (Russia) per 100,000 populations per year [13].

Mortality in the first month after a stroke reaches 35%, and within a year 50% die, and of the survivors, 80-85% remain disabled, only 10-15% return to their usual work activities. Every second stroke survivor needs outside help, which is a nationwide problem [8].

About 85-88% of cerebral strokes are ischemic in nature. Ischemic stroke (IS) in developed countries ranks first as a cause of permanent disability. Movement disorders of varying severity are the most frequent and severe consequences of IS [14]. Hemiparesis in the acute period of IS is observed in 80-90% of cases in 40-50% of patients in combination with sensory impairments [4]. Due to the instability of the vertical posture, functional capabilities are limited and the level of social activity of patients who have undergone IS decreases.

In 30-40% of cases, the genesis of stroke remains unexplained, and in 17% the mechanism of IS cannot be determined even with a comprehensive in-depth examination [11]. According to the classification of E.I. Gusev [9] determine the periods of IS: the most acute - the first 3-5 days from the moment of occurrence; acute - up to 21 days; early recovery - up to 6 months; late recovery - up to two years after stroke. The most acute and acute periods of IS play an important role in the formation and course of an acute ischemic cerebrovascular accident, they determine the severity of IS, and immediate and long-term medical and social outcomes depend on their course [8].

More than 80% of people of working age who have had a stroke become disabled. Movement disorders are the leading factor in disability in a significant number of patients. Movement disorders of varying degrees and nature are the most common symptom of brain damage in both the acute and chronic stages of the disease. In the acute stage, they are detected in 70–90% of patients; after a year, the residual defect persists in at least half of the surviving patients.

The most common, "sign" clinical syndrome of stroke is spastic hemiparesis, traditionally associated with lesions of the pyramidal tracts (pyramidal syndrome). However, motor disorders are more polymorphic and can be associated with direct damage or secondary dysfunction of various parts of a single system of movement regulation, which includes neurons in the motor areas of the cortex, brain stem, basal ganglia and cerebellum, their numerous connections, as well as descending pyramidal and parapyramidal tracts, spinal interneurons and afferent pathways.

Depending on the location and prevalence of the lesion, the phenomenon of diaschisis, concomitant chronic cerebrovascular disease, a complex dynamic constellation of motor syndromes can form, among which the clinician must identify the leading disorder or several disorders that limit the patient's mobility.

The classification of post-stroke movement disorders, given by the outstanding Russian neurologist N.K. Bogolepov in the monograph "Impaired motor functions in vascular lesions of the brain" (1953), in which he distinguished the following variants of motor disorders in stroke:

• Hemiparesis/hemiplegia;
• Changes in reflex activity: protective reflexes, pendulum reflexes, hyperpathic reflexes, cervical tonic reflexes, clonuses;
• Hyperkinesis;
• Friendly movements (synkinesia);
• Contractures.

Nevertheless, now there is a need to supplement it, primarily due to violations of the “higher level” movement, relying, among other things, on the hierarchical theory of the founder of functional neurology, the famous English neurologist H. Jackson. In accordance with this principle, four levels of movement disorders can be distinguished:

1) Movement disorders of the "higher (cortical) level" (apraxia and related disorders);
2) Movement disorders of the "middle level" - pyramidal syndrome, cerebellar ataxia, extrapyramidal syndromes;
3) Movement disorders of the "lower (peripheral) level" (bulbar disorders and other disorders associated with damage to the cranial nerves, contractures and some synkinesis);
4) Combined movement disorders.

Pyramidal disorders are associated with damage to the central motor neurons that make up the pyramidal tract and include negative (pyramidal syndrome proper) and positive manifestations (the so-called "parapyramidal" syndrome) [2]. The pyramidal tract is formed by corticospinal fibers extending from the neurons of all motor areas

of the cortex and heading to the anterior horns of the spinal cord. Approximately 1 million corticospinal fibers pass through each pyramid of the medulla oblongata, but only 30-40% of them are fibers extending from Betz's pyramidal cells in the anterior central gyrus.

Another 30% of the fibers come from the premotor cortex. The remaining 30–40% of the fibers are processes of neurons in the parietal cortex, primarily in the primary somatosensory cortex, which approach the intercalary neurons of the posterior horns and control ascending sensory afferentation and the state of spinal reflexes. Corticospinal fibers, as a rule, have an excitatory effect on spinal neurons, releasing glutamate at their endings, but at the same time they can facilitate both inhibitory and stimulatory mechanisms in the segmental apparatus of the spinal cord [5].

Most of these fibers form synapses directly with the spinal motor neurons of the anterior horns, most often with that part of them that is concentrated in the dorsolateral parts of the anterior horns and innervates the distal parts of the limbs. The fibers that regulate the function of the proximal and axial sections are projected onto the ventromedial part of the anterior horns and more often contact intercalary or propriospinal neurons, indirectly affecting the activity of motoneurons. Through these indirect connections, more massive but less differentiated movements are possible, involving several joints at the same time (for example, stretching the arms and / or walking).

Crossed corticospinal fibers following in the lateral columns preferentially approach the neurons innervating the muscles of the distal extremities, while non-crossed fibers in the anterior columns more often contact the interneurons of both halves of the spinal cord, innervating the muscles of the proximal and axial sections [2] . Comparative studies show that the number of fibers in the pyramidal tracts, as well as the proportion of corticospinal fibers that have direct contacts with motor neurons, is significantly higher in humans than in primates.

This is due to an increase in the complexity of fine differentiated actions that require independent movements of neighboring fingers (for example, when grabbing small objects with a brush). The inability to perform such movements is determined by the non-selectivity of connections between cortical and spinal motor neurons. Most corticospinal neurons are connected by monosynaptic connections to more than one spinal neuron, although the motor field of a given cortical neuron is still limited to a relatively small number of functionally connected muscles (usually 2–3).

Selective damage to the corticospinal tracts (at the level of the pyramids or legs of the brain), following from the anterior central gyrus, causes flaccid paralysis of the limbs, more pronounced in the arms than in the legs. However, then there is a relatively rapid

recovery of the function of the muscles of the trunk and proximal parts of the limbs, later - the function of the distal parts of the limbs, however, fine movements in the distal parts of the limbs, as a rule, remain impaired. Thus, voluntary movements can be carried out without the participation of the corticospinal tract, but in this case they lose their speed, dexterity, and accuracy.

Muscle hypotension gradually regresses, but spasticity and increased tendon reflexes after selective damage to corticospinal fibers from the primary motor cortex do not develop if the following fibers in the pyramidal tract or near it (at the level of the internal capsule, trunk or spinal cord) from the premotor cortex are not damaged, which affect the spinal cord indirectly through the reticulospinal ("parapyramidal") tract (cortico-reticulospinal fibers) [8]. The joint lesion of the corticospinal and cortico-reticulo-spinal fibers largely explains the features of the classic picture of spastic paralysis.

Negative symptoms (symptoms of prolapse) include weakness (inadequate generation of muscle effort) of the extensor and abductor muscles of the arms, as well as leg flexors, loss of selective control over the muscles and segments of the limbs with impaired fine movements, mainly in the distal limbs. Somatotopy of the pyramidal tract (fibers to the arm are located ventromedial, and to the leg - dorsolateral) helps to explain the distribution of paresis. In infratentorial lesions, the paresis more often involves the proximal limb, while the predominant lesion of the distal limb is more often caused by the supratentorial lesion.

Isolated central paresis of the facial muscles is more often explained by damage to the knee of the internal capsule [6]. Weakness of the contralateral limbs without involvement of the face may indicate occlusion of the anterior cerebral artery. The presence of signs of ataxia in the paretic limb (atactic hemiparesis) is possible with a lacunar focus in the bridge or radiant crown. The distribution of paresis may indicate the mechanism of stroke development. For example, a more frequent faciobrachial type of paresis may indicate obstruction of the superficial branch of the middle cerebral artery, while a much rarer monoparesis is more often observed with small cortical infarctions or damage to the semioval center.

“Positive” symptoms include spasticity, revitalization of tendon reflexes (phasic stretch reflexes), the phenomenon of “irradiation” of reflexes, muscle synergies (coactivation of antagonist muscles), pathological reflexes. Forms of muscle hyperactivity are divided into:

• Sensitive to stretching (spasticity, spastic dystonia, spastic synergies);

Insensitive Or Insensitive To Stretching (Extrasegmental Synergies: Synkinesis, Increased Skin (Nociceptive) Reflexes, Muscle Contractions During Autonomic Or Other Reflex Activity (Breathing, Coughing, Yawning)) [12].

The result of muscle hyperactivity may be a change in the biomechanical properties of muscles (stiffness, contracture, fibrosis, atrophy). Although the symptoms of “loss” (paralysis proper) traditionally come to the fore and largely determine the degree of functional defect, the contribution of “irritation” symptoms to disability is often no less significant [6].

The main clinical manifestations of spasticity include:

1. increased resistance to passive movements;
2. the phenomenon of "jackknife";
3. increased tendon reflexes;
4. clonuses;
5. painful muscle spasms;
6. changing walking;
7. persistent shortening of muscles with the development of contracture.

Spasticity is a speed-dependent increase in muscle tone, predominantly involving anti-gravity muscles. As a result, tone predominates in the flexors and pronators of the arm and the extensors and adductors of the leg. With slow passive movements of the forearm and lower leg, no muscle resistance is felt, but with a fast movement, resistance arises, which is quickly overcome (the "jackknife" phenomenon) [11].

Spasticity can impair the function of the affected limbs, making it difficult to carry out voluntary movements and leading to the development of contractures; not have a significant effect on the function of the limbs (paresis or imbalance can be the decisive factor in the motor defect) and even improve motor activity (for example, by giving the limb a supporting function) [14]. Spastic dystonia - tonic muscle contraction with the formation of pathological postures, sensitive to stretching, but not dependent on the speed of movement

Muscle tension in spastic dystonia is caused by supraspinal and spinal mechanisms, persists after deafferentation of the limb, selectively involves agonists and antagonists of various segments of the body, manifests itself not only at rest, but also during movement, leads to dysfunction of the limb, and is accompanied by pain [5]. Muscle contraction decreases with prolonged passive stretching (splinting), blockade with a local anesthetic, and the use of antispastic agents.

Features of gait in spastic paresis caused by damage to the pyramidal tracts are explained by the predominance of the tone of the extensor muscles, as a result of which the leg is extended at the knee and ankle joints and therefore elongated [12].

With hemiparesis, due to the extensor installation of the leg, the patient is forced, bringing the leg forward, to make a swing movement in the form of a semicircle, while the body leans somewhat in the opposite direction (Wernicke-Mann gait). With lower spastic paraparesis, the patient walks slowly, on toes, crossing his legs (due to an increase in the tone of the adductor muscles of the thighs). He has to rock himself to take one step after another.

The gait becomes tense, slow. Gait disturbance in spastic paralysis depends both on the severity of paresis and on the degree of spasticity. The proportion of patients with spasticity during the first month is about 20-30%, and over the next three months' increases to 100% [7]. Depending on the location of the lesion, spasticity can be focal, multifocal, segmental, unilateral, and generalized. The initial severity of paresis is the most significant negative predictor of the recovery of motor functions in general, and in particular, a predictor of the development of spasticity.

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