Introduction

A European Thematic Network to Develop Standardised Measures of Spasticity suggested the following definition: “Spasticity is an interrupted or constant hyperactivity of the skeletal muscles, caused by lesions of the upper motor neuron” [1]. It is characterized by tight or stiff muscles and an inability to control their contractions. In addition, reflex contraction may persist for too long and may be too strong. Spasticity is a physiological result of brain and/or spinal cord injury, which can be life treating or lead to severe disabil-
Morpho-Functional Basis of Spasticity

The motor system consists of neurons and pathways whose integrated activity ensures posture and movements of the body and its individual parts. The upper motor neuron (UMN) controls the speed, strength and direction of the voluntary movements. The cell body (soma) is in the cerebral cortex and its axon extends to the spinal cord (SC) and all the injuries in this section of the pathway are called supranuclear lesions, while the injuries of soma of the lower motor neuron are nuclear lesions [2]. The injuries of axons of the lower motor neuron are called infranuclear lesions. The lower motor neurons of medulla spinalis are involved in many reflex mechanisms.

Of clinical significance for motor activity are myotatic reflex, inverse myotatic reflex and gamma loop reflex, which provide appropriate muscular speed and efficacy [2]. Myotatic reflex is a contraction that occurs in response to stretching within the muscle. Basically, these are annulospiral stretch receptors with Ia afferent fibers, synapse in the SC and an efferent axon of lower α motor neuron that causes muscle contraction. The inverse myotatic reflex starts from the Golgi tendon organs which register an increased tone, with Iβ afferent fibers which cause excitation of spinal interneurons causing inhibition or inactivation of Ia fibers. Gamma motor neurons innervate intrafusal muscle fibers and provide the maintenance of sensitivity of the muscle spindle at the time when muscle is shortened during the contraction.

The muscle tone is permanent, basic muscle tension, which constantly exists but varies in intensity due to muscle activity, action of sensory stimuli of different modality and of current emotional state. It is physiologically controlled by excitatory presynaptic potentials of Ia affrent fibers and inhibitory postsynaptic potentials from muscle spindles of antagonistic muscles. Tone may be altered by hypotonia or hypertonia. The hypertonia in UMNS can be a condition of increased muscle tone (especially of antigravity muscles) that occurs due to the damage or disease of UMN mediated by the stretch reflex or intrinsic, non reflex hypertonia, due to contracture. If the muscle tone is increased due to the lesion of the pyramidal tract, it is called a spasm, and if it occurs due to the damage of extrapyramidal tract, a rigor.

A spasm is a motoric disorder in which an increased muscle tone (especially of antigravity muscles), with slower movements has a tendency of atrophy and/or contractures. It always involves groups of muscles. In spasm, there is an elastic resistance to passive stretching of the muscles and upon the termination of stretching, part of the body returns to its initial position. Sometimes a clonus may occur.

In rigidity, an increased muscle tone is much weaker than in spasm, but the muscles are partly constantly under contraction (especially the antagonists of antigravity muscles). Movements are slow, hypertonia is constant throughout the whole movement, and after the stretching the extremity remains in this newly-established position, i.e. it does not return to its initial position, as with spasm. It involves just one muscle.

The upper motor neuron syndrome represents the lesion of upper motor neuron which leads to the absence of inhibition and to disorder of the reflex arc with spasm. Spasticity, i.e. the hyperactivity of myotatic reflex can be caused by lowering the threshold of excitation of muscle spindles or an abnormal processing of sensory inputs in SC.

Two balanced descending systems are controlling stretch reflex activity: the inhibitory dorsal reticulospinal tract and facilitatory medial reticulospinal tract and vestibulospinal tract. Only the ventromedial bulbar reticular formation, originating from the dorsal reticulospinal tract is under cortical control. Brain damage causes spasticity due to the disruption of facilitatory corticobulbar fibers and causes inhibition of ventromedial reticular formation [3]. Spasticity appears to be caused by loss of reduced excitability of both postsynaptic (decreased reciprocal Ia or Ib inhibition, recurrent inhibition) and presynaptic inhibitory circuits (gamma-aminobutyric acid - GABA-ergic), which control the stretch reflex, postactivation depression decreases at spinal level (independent of the cortex) [4].

Spasticity may be caused by the plasticity of the nervous system. Plasticity of the nervous system is an attempt to restore the function. This tends to promote the formation of new neuronal circuits that allow the formation of new movement patterns by axonal growth and sprouting and an increase in the number of postsynaptic membrane receptors [2, 3]. But the ultimate effect can be excessive, inadequate muscle reflex response to any peripheral stimulation. The overall result is reduced muscle tone and weaker tendon response which is a reason why there is no relaxation and muscle being in constant spasm. New branches toward vestibular, rubrospinal and reticulospinal tract are less selective than pyramidal tract, leading to overactivity. Furthermore, muscle fibrosis and other components of muscle contracture might even increase spasticity.

Spasticity is more often found in flexor muscles of the upper limbs, and in extensor muscles of the lower limbs [1, 3].

There are different degrees of spasticity (depending on velocity and length): the clasp knife phenomenon: increased resistance is present only at the beginning or at the end of the movement; stiffness with resistance throughout the whole passive movement; stiffness with an intermittent resistance to the passive movement [2, 4].
Spasticity can be caused by pain: sensory disturbances, excessive stress on joint and muscles when they are stretched, disruption of some muscle fibers occur and release substances which influence nociceptors. Vs. pain increases spasticity.

The lesion of the lower motor neuron can be caused by trauma or metabolic disorder (alcoholism, diabetes mellitus). A flaccid paralysis appears, hypotonia, pronounced atrophy (70–80%) and only a segment muscles inervated by the damaged alpha motor neuron are affected [2].

Combined upper and lower motor neuron lesions cause so-called alternating hemiplegia, because the deficit of upper motor neuron is manifested contralaterally while of the lower motor neuron - ipsilaterally. This often happens if the lesions are in the brainstem. If the lesions of upper and lower motor neuron are at the level of SC, the symptoms are manifested ipsilaterally [2, 4].

Etiology

Spasticity may be caused by stroke; demyelinating diseases (multiple sclerosis, amyotrophic lateral sclerosis); cerebral palsy; tumors; brain and SC injury (trauma, ischemia, surgical intervention); other neurodegenerative diseases [2–4].

Symptoms

In the UMNS, immediately after the stroke or a trauma, "negative" symptoms appear: weakness, early hypotonia; loss of deep tendon reflexes; loss of dexterity; paresis and paralysis; increased fatigue; pain. Later, other "positive" signs appear: muscle hyperactivity; spasticity; hyper-reflection atettes; spastic dystonia; clonus (series of fast involuntary contractions); cocontraction; abnormal posture, contractures (permanent contraction of the muscle and tendon due to severe persistent stiffness and spasms) and bone and joint deformities. A positive Babinski sign tends to appear soon after the lesion and persists [1–3].

Management of Spasticity: Therapeutic Modalities

Before the treatment begins, it should be considered whether a patient needs a treatment and to what purpose? It is necessary to establish the cause, the degree and distribution of spasticity; localization of injury; comorbidity (contractures, cognitive decline ...); clinical course of the disease; cognitive status of the patient; potential adverse effects; support of the family and community; passive mobility, presence of clonus, reflexes, tonus estimation and active mobility; electromyoneurography; quantitative analysis of walking; spasticity estimation using the Ashworth scale, spasm scale, Wartenberg pendulum test, and Tardieu scale [5, 6].

Sometimes spasticity may also be useful, since it may be: a warning mechanism; preservation of muscle mass; prevention of osteoporosis to a certain extent; maintenance of limb circulation; assistance in rehabilitation process; sometimes ensures stability in cases of muscle weakness – especially for walking; facilitates standing position, transfers and moving with the help of orthoses and aids.

The therapy is justified only if it is associated with intensive pain; with sleep disorders; significantly limited functional capacity of patients; limited capabilities of positioning and orthotising; in prevention of contractures. In patients with UMNS, mobilization of the affected limbs and prevention of prolonged shortened position of affected muscles are probably the most important things to do in order to prevent and treat muscle hypertonia. In this case, physiotherapy is of utmost importance, providing regular and individualized stretching program, along with correct positioning of limbs and application of splints and casts [4].

A multidisciplinary spasticity management includes:

- Prevention: nurture, proper positioning, regular skin inspection and bladder and intestinal program, stretching to maintain the amplitude of movement, prevention of complications such as decubitus, urinary retention, constipation, infection and pain.
- Therapy: physical procedures, work therapy, positioning/ortheses, medications, surgical interventions [3, 5, 6].

The objectives of rehabilitation in spasticity are to reduce muscle tone, maintain or improve the amplitude of movements, increase strength and coordination and improve quality of life; rehabilitation to improve daily life activities; alleviating pain and muscle spasms; utilization of orthoses/splints/trolleys ...; prevention of drug abuse; delay or avoid surgical interventions [7].

Physical therapy and rehabilitation includes kinesitherapy: controlled stretching, muscle strengthening (1h per day, 5 times per week), hydrotherapy, underwater massage, parafango, cryomassage, vibrations, thermoanesthetic modalities, functional electrical stimulation/biofeedback, hippotherapy, neuromuscular facilitation according to the Bobath concept [7–9]. Neuro modulation is also used with the aim of reducing the intensity of neuropathic pain, spasm and spastic pain by: low frequency direct current or injections of medications in the spinal subdural space [3, 10]. Nintendo Wii remote may serve as a convenient and cost-efficient tool for the assessment of spasticity [11].

Medications in the therapy of spasticity may be administered orally, transdermally, via an intrathecal pump, and chemodenervation (blockage). Drugs that are usually used are: medications acting on GABA system: benzodiazepines (diazepam and clonazepam); baclofen; dantrolene sodium; gabapentin; alpha-adrenergic and serotoninergic agents (noradenergetic pathways modulate the presynaptic inhibition of afferent spinal neurons): tizanidine; clonidine; dexmedetomidine; cyproheptadine; central myorelaxants: cyclobenzaprine, carisoprodol, methocarbamol, metaxalone, chlorzoxazone, chlorphenesin, chlorpromazine; and cannabis and cannabinoid-like substances (dronabinol, nabilone). Some medications can be used as mesotherapy [12, 13].
Surgical treatment of spasticity is based on neurolysis, i.e. neurotoxins cause chemodenervation. Local anesthetics can also be used in the removal of spasticity symptoms. Parenteral administration includes: phenol, botulinum toxin, alcohol, lidocaine. The methods of administration are intrathecal drug application or dorsal rhizotomy of lumbar and sacral afferent nerve roots [3, 6, 14].

Conclusion

Spasticity is a complex phenomenon with constant hyperactivity of the skeletal muscles. It is part of the upper motor neuron syndrome and many neurological diseases and disorders. Therapeutic treatment of spasticity should be highly specific, individualized, multidisciplinary and conducted carefully, as well as controlled with the purpose of functional improvement and pain relief. It is of utmost importance to know that sometimes spasticity is useful and should not be removed. There is a need for standardized protocols for ‘good clinical practice’ in the management of spasticity.

References

1. Burridge JH, Wood DE, Hermens HJ, Voerman GE, Johnson GR, van Wijck F, et al. Theoretical and methodological considerations in the measurement of spasticity. Disabil Rehabil. 2005;27(1-2):69-80.
2. Young PA, Young PH, Tolbert DL. Basic clinical neuroscience. 3th ed. Philadelphia: Wolters Kluwer; 2015.
3. Trompetto C, Marinelli L, Mori L, Pelosin E, Currà A, Molfetta L, et al. Pathophysiology of spasticity: implications for neurorehabilitation. Biomed Res Int. 2014;2014:354906.
4. Mirbagheri MM, Settle K, Harvey R, Rymer WZ. Neuromuscular abnormalities associated with spasticity of upper extremity muscles in hemiparetic stroke. J Neurophysiol. 2007;98(2):629-37.
5. Tomasević Todorovic S, Kopčanski S, Mikov A, Bošković K, Petrović, Popović Petrović S, et al. Functional status of patients after stroke. Med Pregl. 2015;68(5-6):181-6.
6. Ansari NN, Naghdī S, Arab TK, Jalaie S. The interrater and intrarater reliability of the Modified Ashworth Scale in the assessment of muscle spasticity: limb and muscle group effect. NeuroRehabilitation. 2008;23(3):231-7.
7. Barak O, Ivetić V, Filipović D, Naumović N, Lukac D, Drapšin M, et al. Event-related potentials following exercise bouts of different intensity. Med Pregl. 2007;60(11-12):531-5. Serbian.
8. Naumović N. Posebna razmatranja u odnosu na lumbalnu radikulopatiju. In: Bošković K, editor. Lumbalna radikulopatija i kvalitet života. 1st ed. Novi Sad: Medicinski fakultet Univerziteta u Novom Sadu; 2009. p. 49-103.
9. Lee YH, Lee YT, Park KH, Kim SH, Jang SM, Kim TH, et al. Effect of EMG-triggered electrical stimulation in patients with chronic hemiplegia. J Korean Acad Rehabil Med. 2003;27(3):320-8.
10. Sommerfeld DK, Eek EU, Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. Stroke. 2004;35(1):134-9.
11. Ward AB. A summary of spasticity management – a treatment algorithm. Europ J Neurol. 2002;9 Suppl 1:48-52.
12. Yeh CH, Hung CY, Wang YH, Hsu WT, Chang YC, Yeh JR, et al. Novel application of a Wii remote to measure spasticity with the pendulum test: proof of concept. Gait Posture. 2016;43:70-5.
13. Naumović N, Bačikin R. Mezoterapija kao komplementarna grana fizikalnoj i rehabilitacionoj medicini: pregled dosadašnjih iskustava. Balneoclimatologija. 2016;40(2):169-74.
14. Smyth MD, Peacock WJ. The surgical treatment of spasticity. Muscle Nerve. 2000;23(2):153-63.