Therapy for Post-Stroke Spasticity

Subjects: Clinical Neurology
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ESWT is safe and free of undesirable side effects. The mechanism of action of ESWT on muscles affected by spasticity is still unknown. To date, no standard parameters of ESWT in post-stroke spasticity regarding intensity, frequency, location, and the number of sessions has been established.

Keywords: stroke ; muscle spasticity ; shock wave therapy ; physiotherapy ; neurorehabilitation ; state of art ; narrative review

1. Introduction

According to a recent paper by Bensmail et al.[1] post-stroke spasticity is estimated to affect up to 43% of stroke survivors and can be seen already in the first week after stroke onset. Spasticity is more common in the upper than lower extremity and is proportional to the severity of upper-limb impairment—the prevalence of post-stroke spasticity increases during the first year[2]. Based on a systematic review of the literature Schinwelski and Sławek [2] found that spasticity within 12 months after stroke develops in 14–44% of patients.

In the Urban et al.[3] study, from 211 patients with ischemic stroke spasticity developed in 41.6% within six months. According to Wissel et al.[4], spasticity manifests itself in 4 to 27% of patients in the first four weeks after stroke onset, in 19 to 26.7% in the second and third month after the disease and in 17 to 42.6% in the phase chronic (>3 months after onset)—the discrepancy of these data results from the difference in the results of observations of different authors. Zorowitz et al.[5] reported that spasticity would develop in about 20–40% of people who survived stroke. According to Yelnik et al. [6] spasticity can reach even 40–70% in the chronic phase of stroke. This brief overview shows how important it is to follow up patients with increased risk of developing spasticity to start adequate treatment and prevent the negative consequences of spasticity.

Since 2005, commonly used is the definition made by experts under the leadership of Pandyan[7]: (Spasticity is) “a disordered sensory-motor control, resulting from an upper motoneuron lesion presenting as intermittent or sustained involuntary activation of muscles”. Severe spasticity may hamper rehabilitation, reduce the functionality, limit the patient’s autonomy, and lead to contractures, pain, and weakness. Spasticity may impair the ability to perform activities of daily living (ADL), household tasks, self-care, may decrease mobility, can cause such symptoms like pain, sleep disturbances, mood changes, depression, anxiety, and may also reduce patient’s quality of life and cause a negative impact on family and caregiver’s relationship[5][8].

2. Development of Post-Stroke Spasticity

Spasticity, a neurological impairment, is a common but inevitable consequence of an upper motor neuron (UMN) syndrome. It is one of many sensory-motor signs and symptoms that may be present following an UMN lesion [7]. Lesions involving both the pyramidal and parapyramidal pathways cause increased excitability of alpha-neurons in skeletal muscle.

It is hard to predict the development of spasticity after stroke. Kong et al.[9]evaluated the occurrence and temporal evolution of UL spasticity in 163 patients with a first-ever ischemic stroke (NIHSS 8.5 ± 5.2, UEMI 35.8 ± 30.8, mean age 63.8 ± 10.7 years) admitted to a rehabilitation unit. Spasticity of UL occurred in 33% of patients at three months after stroke. The development of spasticity at later stages of the stroke was infrequent, occurring in only 17%. In patients with mild spasticity (Ashworth Scale score 1) at three months after stroke, the worsening of spasticity occurred in only one patient.

On the other hand, almost half of the patients with moderate spasticity (Ashworth Scale score 2) at three months progressed to severe spasticity (Ashworth Scale score 3). Reduced UL activity was the most important correlate of “moderate to severe spasticity” (Ashworth Scale score ≥ 2) (p < 0.001), and poor UL strength on admission to

rehabilitation, the most important predictor of “moderate to severe spasticity” ($p < 0.001$). In conclusion: Selective monitoring to detect severe spasticity is recommended for patients with an Ashworth Scale score of two or greater at three months after stroke, and in patients with severe UL weakness on admission to rehabilitation[9].

Lundström et al.[10] stated that the prevalence of any spasticity 12 months after first-ever stroke was 17% and of disabling spasticity 4%. Patients with DS scored significantly worse than those with no disabling spasticity (DS) on the Modified Rankin Scale (MRS) ($p = 0.009$) and the BI ($p = 0.005$). Disabling spasticity was more frequent in the upper extremity. It positively correlated with other indices of motor impairment and inversely with age. There was an independent effect of severe upper extremity paresis and age below 65 years. Opheim et al.[11] evaluated the upper-limb (UL) spasticity during the first year in 117 patients after the first-ever stroke. Spasticity was present in 25% of the patients at day 3 and 46% at 12 months. In most patients with spasticity, the severity increased during the first year after stroke. Spasticity appeared first in the elbow flexors and later in the elbow extensors and the wrist flexors. The patients with spasticity had a significantly worse sensorimotor function, and more pain reduced joint ROM, and reduced sensibility.

Sommerfeld and et al. [12] assessed the occurrence of spasticity after first-ever stroke (SSS 0–18, 35 men, the mean age of 78 ± 9.5 years) in 95 patients and its association with motor impairments and activity limitations. Spasticity was present in 19% of the patients investigated three months after stroke. Severe disabilities were seen in almost the same number pf patients with or without spasticity.

Wissel et al.[13] based on a literature survey, stated in 2015 that post-stroke spasticity could be a key reason for patients failing to meet physiotherapy goals.

3. Physical Therapy for Post-Stroke Spasticity

In the last decades, various non-pharmacological interventions have been described in managing spasticity in various neurological conditions. They are used as an adjunct therapy to conventional routine care (pharmacological and rehabilitation)[14].

There is a wide range of well-evidenced neurorehabilitation methods used along with pharmacological agents for management of spastic muscle and improvement of motor recovery after stroke. A common neurophysiological concept is neurodevelopmental treatment by Bobath (NDT) and proprioceptive neuromuscular facilitation by Kabat and Knot (PNF) [15][16][17].

We can include such rehabilitation exercise methods, such as constraint-induced movement therapy (CIMT), sensorimotor movement training (SMT), task-related training (TRT), robot-assisted training (RAT), whole body vibration training (WBVT), mirror visual feedback training (MVFT), electromyographic biofeedback training (EMG-BTF), and virtual reality-based training (VRBT). Moreover, there physical therapy agents can be also used, such as neuromuscular electrical stimulation (NMES), electromyography neuromuscular electrical stimulation (EMG-NMES), transcutaneous electrical nerve stimulation (TENS), functional electrical stimulation (FES), therapeutic ultrasound (TU), as well as cryotherapy (cold water, ice packs, and evaporative sprays), thermotherapy (hot water, sauna, and infrared heat), hydrotherapy, and acupuncture[15][18][19].

Neuromodulative methods, such as repetitive transcranial magnetic stimulation (rTMS) or transcranial direct current stimulation (tDCS), are quite promising to be useful in reducing spasticity after a stroke, but still need to be confirmed by larger and multicenter randomized controlled trials to provide evidence of effectiveness under different neurological conditions[20][21][22].

Despite the available range of non-pharmacological interventions for spasticity, there is a lack of high-quality evidence for many modalities. Khan et al.[14] included 18 systematic reviews to evaluate the evidence for a range of non-pharmacological interventions currently used in managing spasticity in various neurological conditions. There is “moderate” evidence for NMES and acupuncture as an adjunct therapy to conventional routine care (pharmacological and rehabilitation) in persons following stroke. “Low” quality evidence for rehabilitation programs targeting spasticity (such as CIMT, stretching, dynamic elbow-splinting, extracorporeal shock therapy (ESWT) in brain injury; tDCS in stroke; rTMS and TENS for other neurological conditions; and physical activity programs, WBVT, and stretching for other neurological condition. For other interventions, evidence was inconclusive.

Over the last 20 years, especially during the last 5 years due to the intensification of research in this area, it has become proven that the ESWT procedure is a safe and effective alternative method to reduce muscle spasticity in post-stroke stroke patients (but also in spasticity related to cerebral palsy, multiple sclerosis, spinal cord injuries, and brain injuries) as a valuable adjuvant modality to standard treatment and rehabilitation[23].

All experts agree that ESWT could not be the only method of treatment spasticity. A short wave can only support comprehensive rehabilitation. Reviewing the multidisciplinary rehabilitation following botulinum toxin and other focal intramuscular treatment for post-stroke spasticity Demetrios et al.[24] stated that the optimal types (modalities, therapy approaches, and settings) and intensities of therapy for improving activity (active and passive function) in adults with post-stroke spasticity, in the short and longer-term, are unclear. Further research is required to build evidence in this area.

4. Shock Waves for Post-Stroke Spasticity

According to Wang et al.[25] extracorporeal shock wave therapy (ESWT) is used since 1982 for lithotripsy to treat kidney stones, urinary calculi, and biliary calculi using an acoustic pulse. It is also reported to be used for salivary stones and pancreatic stones. ESWT is a non-invasive treatment that involves creating a series of low energy acoustic wave pulsations that are directly applied to an injury through a person's skin via a gel medium. There are three types of ESWT: fSWT (focused shock wave therapy), rSWT (radial shock wave therapy), or pSWT (planar shock wave therapy). Recently rSWT in connection with fSWT is mostly used. In the past 15 years, ESWT had emerged as the leading choice in the treatment of many orthopedic disorders, including proximal plantar fasciitis of the heel, lateral epicondylitis of the elbow, calcific tendinitis of the shoulder, and non-union of long bone fracture. Many different parameters of ESWT are used such as energy flux density (EFD): 0.01–0.5 mJ/mm^2, pressure: 10–100 MPa, number of pulses: 1200–4000, and frequency: 1–12 Hz. This enables the penetration of shock waves from 3 cm up to 12.5 cm depth (theoretically). ESWT is widely used for acute and chronic musculoskeletal disorders including low back pain[26][27].

According to the findings of basic sciences research, the ESWT has been shown to promote the activation of a number of molecular and immunological reactions resulting in improved blood circulation, stimulating angiogenesis and neovascularization reactions as well as activating anti-inflammatory responses. Moreover, strong regenerative properties of ESWT towards increased fibroblast recruitment and reduced tissue apoptosis have been observed[28]. Moreover, it was proven that ESWT increases the activation of the vascular endothelial growth factor (VEGF) and its neuroprotective properties as well as the expression of neurotrophin-3 (NTH-3) which improves the neuroregenerative processes. In addition, the ESWT stimulates neurogenesis by increasing proliferation of neural stem cell (NSC), which can improve the functioning of the nervous system[29]. The mechanisms of ESWT action on spasticity reduction are still under investigated; however, there are a few hypotheses, which attempt to explain these mechanisms. First of all, it is being suggested that ESWT is responsible for inducing nitric oxide (NO) synthesis, which is responsible for the formation of new neuromuscular junctions. Another hypothesis concerns the reduction of excitability of motor neurons by the production of continuous or intermittent pressure on the tendons by the ESWT. It is also suspected that ESWT shows antispastic effects by temporarily disturbing neuromuscular transmission in terms of reducing acetylcholine receptors in neuromuscular junctions[30].

There is also a division according to which there are common mechanisms of biological action for fSWT and rSWT, such as increasing the permeability of cell walls, stimulation of microcirculation (of the blood and lymphatic system), and release of substance P (SP) as a neurotransmitter involved in a multitude of neuronal signaling pathways and responsible for pain modulation. However, there are also mechanisms that only characterize fSWT, such as cavitation, release of nitric oxide (NO) responsible for increased cellular metabolism, neovascularization, angiogenesis, and anti-inflammatory effects, as well as stimulation of growth factors, e.g., fibroblast growth factors (FGF) and transforming growth factor (TGF) [31]. Moreover, the theory of mechanotransduction as a biological pathway explaining a cellular effect should be mentioned in the context of biological mechanisms of ESWT action. Mechanobiology is a quite new branch of biological sciences and the ESWT as the mechanical stimulation (mechanotherapy) has a special place and it is under research interest[31][32].

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