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Neurorehabilitation in spasticity

Neurorehabilitacja w spastyczności

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SUMMARY. ***Aim.*** *There are different therapuetic approaches to the neurorehabilitation of spasticity which will be addressed in this paper.* ***Review.*** *The term "spasticity" is used for velocity-dependent muscle tone increase, but also for enhanced tendon reflexes with positive Babinski's sign, exaggerated cutaneous and autonomic reflexes, involuntary flexor and extensor spasms, clonus, impaired movement execution, enhanced muscular resistance against passive movement, abnormal limb postures, clumsiness, enhanced fatigability and paresis. The definition of the term spasticity has changed in the last decades. First it referred only to the velocity-dependent increase in muscle tone, more recently all the above-mentioned phenomena are summarized as the upper motor neurone syndrome. These different definitions serve not an academic purpose but are of practical importance. Most pharmacological studies dealing with drug treatment of spasticity use stretch reflex excitability and/or clonus intensity as parameters to assess the efficacy of antispastic treatment. Velocity-dependent muscle tone and enhanced stretch reflexes have only little functional significance for the patient as the intensity of stretch reflex responses and of clonus hardly corresponds to the quality of movement execution. Dyssynergic patterns of muscle activation with co-activation of agonists and antagonists, paresis and loss of dexterity together with fatigability are much more important for the functional outcome. Increased muscle tone has a potential value. Extensor hypertonicity can provide the rigidity for weight-bearing stance. Spasticity should be treated however if it interferes with nursing, contributes to contractures, causes painful spasms or hampers movements.* ***Conclusions.*** *The first step is to exclude any noxious and enhancing stimulus like urinary tract infections, pain, bowel and bladder distensions, pressure sores etc. Physiotherapy, physical stimuli (ice, electrical stimulation), pharmacotherapy, chemical blocks or neurosurgical interventions should be selected or combined according to the individual status of the patient.*

STRESZCZENIE. ***Cel.*** *Przedstawienie różnych zagadnień terapeutycznych istotnych w neurorehabilitacji spastyczności.* ***Poglądy.*** *Termin „spastyczność" jest używany dla określenia wzrostu napięcia mięśnia zależnego od szybkości rozciągania, lecz cechuje się także wzmożeniem odruchów rozciągowych z dodatnim objawem Babińskiego, wygórowaniem odruchów skórnych i wegetatywnych, mimowolnymi skurczami zginaczy i prostowników, klonusami, ograniczeniem wykonywania ruchów, wzmożonym oporem przeciw ruchom biernym, nieprawidłowym ułożeniem kończyn, niezdarnością, wzmożoną męczliwością i niedowładami. Definicja spastyczności zmieniła się w ciągu ostatnich dekad. Z początku odnosiła się jedynie do zależnego od szybkości (rozciągania) wzrostu napięcia mięśniowego, w późniejszym okresie wszystkie wymienione powyżej zjawiska zostały zsumowane jako zespół górnego motoneuronu. Te zróżnicowane definicje nie służą jedynie celom czysto akademickim lecz są ważne w praktyce. Większość badań nad leczeniem farmakologicznym spastyczności używa pobudliwości odruchu rozciągania i/lub nasilenia klonusów jako parametrów oceny skuteczności leczenia przeciwspastycznego. Zależne od szybkości (rozciągania) napięcie mięśniowe i wzmożone odruchy rozciągowe mają jedynie małą czynnościową znamienność dla pacjenta. Intensywność odruchów rozciągowych i klonusy mało korespondują z jakością wykonywania ruchów. Dyssynergiczne wzorce aktywacji mięśni z koaktywacją agonistów i antagonistów, niedowłady i utrata sprawności wraz z męczliwością są dla efektu funkcjonalnego ważniejsze. Wzmożone napięcie mięśni posiada swoje potencjalne zalety. Hypertonia prostowników może zapewnić sztywność niezbędną podczas stania. Spastyczność powinna jednak podlegać leczeniu jeżeli utrudnia pielęgnację, prowadzi do przykurczów,*

powoduje bolesne spazmy lub krępuje ruchy. ***Wnioski.*** *Pierwszym krokiem jest wykluczenie takich szkodliwych i wzmagających napięcie bodźców, jak: infekcja dróg moczowych, bóle, rozciągnięcie pęcherza moczowego i odbytnicy, odleżyny itp. Wybór fizjoterapii, bodźców fizycznych (lód, stymulacja elektryczna), farmakoterapii, blokad chemicznych czy interwencji neurochirurgicznej powinien być dokonany indywidualnie, w zależności od stanu pacjenta.*

Key words: spasticity / neurorehabilitation
Słowa kluczowe: spastyczność / neurorehabilitacja

ASSESSMENT OF SPASTICITY

Spasticity can be assessed by clinical, biomechanical and electrophysiologic measures although these measures indicate little about the degree to which the increased tone affects motor performance. A commonly used clinical scale of spasticity is the Ashworth Scale with levels ranging from 0 (no increase in tone) to 5 (affected joint fixed rigidly).

Ashworth Scale

1. no increase in muscle tone.
2. slight increase in muscle tone, mild clasp-knife phenomenon, or minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension.
3. slight increase in muscle tone, clasp-knife phenomenon, followed by minimal resistance throughout the remainder (less than half) of the range of motion.
4. more marked increase in muscle tone through most of the range of motion, but affected part(s) easily moved.
5. considerable increase in muscle tone, passive movement difficult.
6. affected part(s) rigid in flexion or extension.

The clinical Ashworth Scale has a good interrater variability and reproducibility. The Wartenberg's pendulum test is a biomechanical technique during with tester lifts the relaxed leg and releases it so the leg swings by gravity. Knee ankle, velocity and number of swings are measured with goniometers. More complicated biomechanical methods involve torque motors to measure the torque when a joint is moved.

PHYSIOTHERAPY: SUSTAINED MUSCLE STRETCH

Spasticity reduction is achieved according to the Bobath concept [1] by stretching the spastic muscles slowly and tonically in a proximo-distal sequence beginning with a prolonged outward scapula rotation. Then the arm is abducted and rotated outward in the shoulder and extended in the elbow, followed by an extension in the wrist and finger joints and an abduction of the thumb. In the position in which most spastic flexor muscles were tonically stretched, muscle tone gradually decreased. Normally after 10–15 min reduction in muscle tone had reached its maximum. Besides the manual muscle stretch performed by the physiotherapist, another method for obtaining an effective reduction in the spastic muscle hypertonus is to keep the wrist and the finger joints in an extended position by means of distal splinting or plaster casting [2, 3, 4].

Since spasticity (as well as raised stretch reflex excitability) can be explained at least in part by enhanced motoneurone excitability [5] the effect of spasticity-reducing physiotherapeutic interventions can be measured as reduced excitability of alpha-motoneurones by transcranial magnetic stimulation (TMS). The physiotherapeutic method of sustained stretch of spastic muscle groups exerts an in-

hibitory effect on spinal a-motoneurones that is reflected in the response characteristics to TMS. TMS after 24–48 h of casting elicites similar modifications in muscular response characteristics as after the physiotherapeutic stretching intervention. Distal casting for about 48 h had a clearly longer lasting effect in comparison to physiotherapeutic spasticity reduction by muscle stretch for 10–15 min.

Sustained muscle stretch as proposed by Bobath [1] is able to reduce enhanced motoneuronal excitability. The effect is mediated by stretch receptor adaptation to the new extended position with a relatively reduced excitatory drive from the muscle spindles when the hand is returned to a slightly flexed position. The same model of receptor adaptation or fatigue holds true for distal casting with the additional effect of a longer lasting spasticity reduction. An inhibition of the enhanced tone of the stretched muscle by Golgi tendon organs is less probable, as these respond predominantly to actively generated tension. Nevertheless, Ib-inhibitory interneuroneurones known to mediate input from Golgi tendon organs onto the alpha-motoneurones of homologous muscles are assumed to play an important role in transmitting inhibitory influences [6, 7] arising in even distant muscles being stretched by the physiotherapist at the beginning of the described proximodistal stretching sequence. It is a common observation that the spastic hypertonus of distal flexor muscles lessens as soon as the stretching sequence starts in the shoulder and proximal arm musculature. This transmission of inhibition is thought to be mediated via Ib-inhibitory interneurones serving as an integrating relay station of influences from muscle spindle afferents arising in more proximal muscle groups. The aforementioned mild inhibitory effect on extensor motoneurones may also be explained by this mechanism.

In summary, the enhanced excitability of spinal amotoneurones and interneurones in the spastic state is reduced by sustained muscle stretch starting in the shoulder and proximal arm musculature. The inhibitory effect of this physiotherapeutic intervention can be explained by stretch receptor adaptation in the spastic target muscle and by an inhibition due to early stretching of more proximal muscles mediated via the integrating Ib-inhibitory interneurones projecting to the target muscle via intersegmental propriospinal interneurones [8].

SPASTICITY REDUCTION BY VOLUNTARY MOVEMENTS

The therapeutic strategy of Bobath [1] always starts with the reduction in spasticity. The theory proposed by Bobath that strong recurrent Ia inhibition from the spastic muscle forms the principal basis for the weakness of its antagonist has not been confirmed. Ia inhibition is known, instead, to be less effective in hemiparetic patients [5]. Therefore, spasticity reduction by sustained stretch does not deliver a powerful disinhibition of the alpha-motoeurones of the weak antagonist. Probably the most important effect of sustained stretch for the release of voluntary movements is mechanical in nature. In the case of spasticity in hand and finger flexors voluntary movements of the weak hand and finger extensors could become possible if the antagonistic flexor hypertonus is reduced sufficiently to allow relatively free extension movements.

One decisive drawback of the Bobath method is the aforementioned avoidance of voluntary active movements in weak muscle groups before maximal reduction of muscle tone in spastic muscle groups has been achieved.

Recent experiments in our laboratory aiming at reinforcing and training rapid voluntary hand and finger movements [9, 10] in patients with a central paresis of the hand showed that the frequency of associated reactions and the spastic hypertonus of hand and finger flexors diminished (as expressed on the Ashworth scale) during the training phase of several weeks while hand and finger

function improved. It was also shown that emg-initiated stimulation of muscles and repetitive training improved function and decreased spasticity [10]. It can be hypothesized that the defective Ia inhibition from the weak extensors on the flexors becomes more effective parallel to the improvement in hand and finger function.

The occurrence of "associated reactions" is a frequent phenomenon in the physiotherapeutic method of Brunnstrom [11]. She emphasizes the use of central facilitation techniques, i.e., maximal innervation of corresponding muscles on the intact side of the body or innervation of less weak proximal muscles of the affected extremity to recruit more paretic distal muscle groups by overflow or irradiation mechanisms. In this context synergistic movements and the development of higher muscle tone are often observed. Nevertheless, Brunnstrom recognizes the necessity of refining the patient's movement repertoire [11]. She therefore utilizes localized skin stimulation and tapping over the muscle belly to facilitate the activity of individual muscles or of distinct muscle groups. Her argument that facilitation of circumscribed muscle groups is appropriate to diminish spasticity of their antagonists via Ia inhibition does not take into account that Ia inhibition is defective in patients with central hemiparesis [5]. Cheney et al. [12] described a reciprocal inhibition of corticomotoneuronal cells on antagonist muscles during voluntary innervation of the agonist in awake monkeys. Whether suppression of antagonist activity during agonist innervation at the cortical level also takes place in hemiparetic patients must be doubted [9]. Nevertheless, it may be assumed that inhibition of spastic antagonists by Ia inhibition increases parallel to the improved voluntary movement capacity in the agonist.

Comparable mechanisms of spasticity reduction by reciprocal Ia inhibition are used by therapists applying proprioceptive neuromuscular facilitation (PNF) techniques [13]. Synergistic coactivation within particular multijoint movement patterns is the basic principle in the PNF technique, using irradiation effects from less paretic proximal muscles to produce and strengthen contraction in weak distal muscles. Parallel to the increase in strength of the target muscle group, la inhibition is thought to inhibit spasticity in the respective antagonistic muscle group. In clinical practice, however, the occurrence of associated reactions and a tendency toward undesired spastic limb postures is a common observation during PNF therapy of stroke patients with severe hemiparesis.

MECHANICAL FACTORS IN SPASTICITY ARE OFTEN UNDERESTIMATED

Apart from the neuronal mechanisms, mechanical factors concerning the viscoelastic and contractile properties of the muscle must not be underestimated [14]. Muscle stiffness is determined by elasticity of the contractile elements and the connective tissue as well as by the discharge frequence of the motor. Discharge frequency is influenced by sustained stretch. Nevertheless, as physiotherapeutic techniques adhere most frequently to neurophysiological principles, it is important to stress the necessity of daily passive range-of-motion exercises to prevent, at least in part, an increase in muscle stiffness and a joint and tendon tightening.

Stroke patients who spend a prolonged period in the wheel chair show a tendency to develop a marked imbalance in muscle tone in the affected lower extremity toward hip and knee flexion. This undesired flexion in hip and knee together with ankle plantar flexion may be overcome by placing the body weight on the affected joints while the patient stands with the affected joints in the extended position. The same effect, i.e., prolonged stretching of tight muscles, can be achieved by putting a wedge between the limbs that tend to approach each other. This method is most appropriate for treating leg scissoring due to hip adductor spasticity in paraplegics.

Prolonged tonic stretching of circumscribed spastic muscles or muscle groups can also be achieved by static and/or dynamic splints [3, 4, 15], the aforementioned distal casting, or special orthoses adapted to the individual patient, for instance, ankle-foot orthoses or knee-ankle-foot orthoses [16].

MUSCLE COOLING TO PREPARE PHYSIOTHERAPY

Muscle cooling is of benefit for the spastic patient provided that it is applied long enough. This influences particularly stretch reflex activity and clonus. The initial effect of cold application is even facilitatory, as has been described by Rood [17]. Hagbarth [18] demonstrated that phasic localized skin stimulation by „icing" induces an enhanced gamma-innervation of the muscles underlying the stimulated skin. This in turn causes increased sensitivity in stretch receptors with increased stretch response and enhanced muscle tone.

The administration of prolonged and deep muscle cooling leads in many cases to a sufficient relaxation in spastic muscle groups. PNF therapists were the first to use penetrating muscle cooling in preparation for their active movement therapy to reduce muscle tone and to eliminate pain.

The almost immediate drop in stretch reflex excitability following the administration of cold cannot be due to intramuscular temperature reduction since muscle temperature requires several minutes (depending on the muscle mass) to fall significantly. The effect must therefore be mediated by a decrease in skin receptor sensitivity and its influence on alpha/gamma-motoneurone. Surprisingly, clonus is absent only when intramuscular temperature is markedly. It is unlikely that clonus is abolished due to transmission failures or blocks caused by cooling of the motor nerve since M and H responses show only moderate increases in latency. Unfortunately, the effect of cold on muscle spindle sensitivity has not been sufficiently clarified.

Animal experiments are in part contradictory. Eldred et al. [19] described a nearly linear decrease in muscle spindle sensitivity parallel to reduction in muscle temperature, whereas Lippold et al. [20] found an enhanced spontaneous firing rate at muscle temperatures 2–3°C below body temperature. Nevertheless, studies in humans describe intramuscular temperatures of 4–5°C below body temperature so that muscle spindle discharge is considered to be reduced. Whether the proposed effect is exerted exclusively on the muscle spindle itself or is the result of a suppression of the gamma-innervation has not been clarified up to now. In addition to the neuronal mechanisms, the reduction in muscular hypertonus can also be caused by altered viscoelastic and/or contractile properties of the muscle. Miglietta [21] found markedly prolonged muscle contraction times after deep muscular cooling. Spasticity reduction by deep muscle cooling is of increasing significance in various physiotherapeutic concepts since it allows the beginning of an active functional neuromuscular training of the weak agonists during the period of cold blockage of the spastic antagonist.

ELECTRICAL AND MAGNETIC STIMULATION

Electrical stimulation of motor and sensory nerves, muscles, and dermatomes by a variety of paradigms has, in general, reduced spasticity at the ankle, knee and wrist. A single stimulation session decreases resistance and clonus for a few hours. Studies of long-term use show a range of responses that, in part, result from variations in patient characteristics, outcome measures, location of the stimulation, and parameters of the electrical stimuli. One study of patients with chronic stroke spastic hemiparesis who had showed that 15 daily, low-intensity, high frequency transcutaneous electrical nerve stimulation (TNS) applications for 1 hour over the proximal common peroneal nerve decreased

a clinical measure of spasticity, increased vibratory inhibition of the H-reflex of the soleus muscle, improved voluntary dorsiflexion force, and reduced the magnitude of the stretch reflex in the affected ankle. Enhanced presynaptic inhibition was considered a contributing mechanism.

Stimulation of FRAs by peroneal and sural nerve stimulation has also been suggested as the cause of similar positive results in patients with myelopathies. Electrostimulation for 5 to 10 minutes by a rectal probe to elicit ejaculation had the added effect of reducing muscle spasms and tonus in 10 of 14 patients for about 9 hours. Changes in muscle tone have not been systematically reported during functional electrical stimulation (FES) studies of muscle in which the primary aim is to increase muscle mass, improve conditioning, or assist ambulation, but some patients with SCI report less spasticity. Electrical stimulation of the forearm muscles can at least transiently reduce flexor tone in the hand. Moreover, a metal mesh glove that conducts electrical impulses has decreased finger flexor postures in the hemiplegic upper extremity in some patients. The subjective measures of spasticity, the variations in stimulation techniques, and the lack of a control therapy during trials.

A new interesting method is the application of the repetitive magnetic stimulation of the spinal cord. Nielsen et al. [22] applied this technique in 12 spastic multiple sclerosis patients. The coil was placed at the mid thoracic 2 level, and within a cycle of 30s, the stimulation (intensity 45–60% of maximum stimulator intensity, rate 12 Hz) was on 8 s; the whole session lasted 30 min. One day later, the patients reported a reduction of leg muscle tone, a finding which was supported by torque and reflex measurements.

Struppler et al. [23] applied the same technique in the treatment of upper limb spasticity in stroke patients. These authors, however, stimulated motor points of selected upper extremity muscles. After two series of 30–50 cycles (maximum rate 40 Hz, motor threshold intensity), each of induced extension and flexion movements of hand and fingers, the patients could extend the paretic fingers with larger displacement amplitude and diminished flexor spasticity for several days. As an explanation, the authors discussed both inhibitory and facilitatory mechanisms, as well as an increased proprioceptive drive by the repetitive magnetic stimulation. Emg-initiated electrical stimulation together with repetitive training in hemiparetics improved function and decrease spasticity [10].

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