



Rehabilitation procedures in the management of spasticity

N. SMANIA¹, A. PICELLI¹, D. MUNARI¹, C. GEROIN¹, P. IANES¹, A. WALDNER², M. GANDOLFI¹

Spasticity is a major disabling symptom in many patients with spinal and/or cerebral lesions. During functional movements, spasticity manifests itself within the complex condition of the “spastic movement disorder”. The pathophysiology of the spastic movement disorder relies on multiple factors including abnormal supraspinal drive, abnormal control of reflex activities, and changes in muscle mechanical properties. The most widely used procedures for management of spasticity are represented by pharmacological treatment aimed at inhibiting reflex hyperexcitability. In the last decades, several non pharmacological procedures for treating spasticity have been put forward, including muscle stretching, muscle reinforcement, physical agents and pain management. These procedures may have both neurophysiological and biomechanical effects on the spastic movement disorder. In the present paper, the literature concerning non-pharmacological procedures in the treatment of spasticity was reviewed and discussed, taking into account the multifaceted pathophysiology of the spastic movement disorder. Although

¹Neuromotor and Cognitive Rehabilitation Research Centre, Department of Neurological, Neuropsychological, Morphological and Movement Sciences University of Verona, Verona, Italy
²“Villa Melitta” Rehabilitation Clinic, Bolzano, Italy

further research in this field is recommended, existing evidence supports the potential role of rehabilitation interventions as a therapeutic tool, which could be integrated with traditional pharmacological procedures in the management of the spastic movement disorder.

KEY WORDS: 'Muscle spasticity - Muscle stretching exercises - Muscle strength - Pain.

Spasticity is a common symptom seen in many neurological conditions (stroke, multiple sclerosis, spinal cord injury, traumatic brain injury and other central nervous system lesions). It has been defined as a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflexes (muscle tone) with exaggerated tendon jerks.¹

This definition has greatly influenced the evaluation and treatment of spasticity for many decades. Based on this definition, spasticity assessment has typically focused on evaluating levels of increase in stretch and tendon reflexes, while spasticity treatment has been directed at reducing reflex hyperexcitability. However, the above definition of spasticity describes phenomena seen in patients while they are resting, and consequently assessment does not take into account its impact on functional movements.

Acknowledgments.—Authors wish to thank Valentina Varalta for her help in reviewing the paper.

Fundings.—A Grant from the CariVerona Fondation (PACIS), Verona-Italy, supported the study.

Corresponding author: N. Smania, Neuromotor and Cognitive Rehabilitation Research Centre, Department of Neurological, Neuropsychological, Morphological and Movement Sciences, University of Verona, Italy. Via L.A. Scuro 10, 37134 Verona, Italy. E-mail: nicola.smania@univr.it

It is now becoming clearer to researchers that during functional movements, such as walking, reflex hyperexcitability manifests itself within the complex clinical condition of spastic movement disorder (SMD).² This condition does not only refer to spasticity, but also to the effects of other associated impairments (weakness, increased muscle stiffness, etc.) on movement patterns. Thus, SMD represents the different determinants of pathological movement patterns in patients with spasticity in a more comprehensive way.²

The main causes of SMD are hyperexcitability of short-latency reflexes, reduction or loss of long-latency reflexes, abnormal supraspinal drive with decrease in maximal voluntary muscle power and altered mechanical muscle properties.^{2,3} Other conditions, such as pain, prolonged altered postures and limb non-use have been also considered as important factors that may negatively influence the severity of SMD.³

In spite of the multifaceted aspects of the SMD condition, treatment currently used in clinical practice is primarily directed at to the reduction of reflex hyperexcitability, by means of pharmacological interventions aimed at treating the so called generalized, regional or focal spasticity.⁴

It is worth noting that in recent years approaches aimed at improving SMD's disabling manifestations, such as difficulty in upper limb movements or gait impairments, have been put forward. These interventions, which include muscle stretching procedures, muscle strengthening exercises, physical agents treatment (shock waves, ultrasounds, electrical stimulation etc.) and pain management, could be combined with classical pharmacological interventions in order to approach the disorder in a more complete, and probably more effective way.

The aim of the present study is to review the literature dealing with non-pharmacological interventions for treatment of spasticity in order to give an up-to-date report regarding the usefulness of the most commonly used procedures. The clinical impact of these interventions will be discussed taking into account the complex framework of the SMD.

Muscle stretching

Muscle stretching, a very popular exercise approach in athletic training programs,⁵ is primarily

aimed at improving the viscoelastic properties of the muscle-tendon unit in order to reduce the risk of muscle-tendon injury.

Nowadays, this approach is becoming a very common practice in the rehabilitative management of SMD. It includes several types of muscle elongation procedures⁶ that can be applied by moving the joint through the range of motion (ROM) manually, or by means of different mechanical devices, to normalize muscle tone, maintain or increase soft-tissue extensibility, reduce contracture pain, and improve motor function.^{6,7} Stretching can increase the extensibility of soft tissues by a mechanism that involves viscous deformation and structural adaptations of muscle and other soft tissues. Structures that are put under tension can consist of muscle, tendon, connective, vascular, dermal and neural tissue.^{6,8}

Stretching exercises can be executed in a very large number of modalities,⁶ which include: 1) passive stretching (the stretch is performed by another person and the patient does not actively participate); 2) active stretching⁹ (the patient initiates and/or maintains the stretch); 3) prolonged positioning¹⁰ (positioning is used to achieve a longer duration stretch of a particular muscle or muscle group); 4) isotonic stretching¹¹ (the limb is moved slowly to its maximum ROM and then held in that position for a that variable length of time); and 5) isokinetic stretching¹¹ (the limb is moved continuously with a force at a constant angular velocity, and thus the stretched position is not maintained).

Regardless of the stretching modality, different features have been defined. The intensity of the stretch is the amount of tension that is applied to the structure(s), which can differ in the amount of force, and be kept either constant or varied.¹² The velocity of the stretch is the speed at which the elongation is occurring. Repetitions are the number of replications of the stretch within one single session. The duration is the time the structures are elongated within one repetition.¹³ The dose is the total end range time; in other words, the total time structures are elongated. Finally, the frequency is the periodicity of the stretch, which can vary from a single session to daily sessions for several weeks.

All of the above mentioned features are often used in conjunction with other interventions, such as splints and orthoses, casting, surgery, or spasticity reducing medications.¹⁰

Of these interventions, casting is becoming a widely used technique which could provide prolonged muscle stretching. It consists of immobilizing the affected limb in a predetermined position by means of moulded casts made of either plasters or tape materials.

This technique is usually used in both upper and lower limbs to improve muscle length, increase joint ROM, and achieve reductions in contracture, pain and spasticity.

Although this technique is widely used in rehabilitation, there is no evidence-based guideline for its use. Thus, the decision to use casting is based on clinical opinion and habit rather than on scientific evidence.¹⁴

Despite this lack of scientific evidence, three main theoretical rationales can be identified for applying a cast: 1) the neurophysiologic rationale considers that casts may reduce excitatory input of muscle spindles, preventing changes in muscle length and thus reducing spasticity; 2) the biomechanical rationale considers that casting can prevent and reduce contractures; 3) the motor learning rationale proposes that casting provides adequate support to proximal joints until sufficient control is gained distally.¹⁴

Each rationale has a reasonable chance of being correct, as scant work has been done to test them.

Only a few high quality studies have focused on evaluating the effectiveness of upper or lower limb casting. They have demonstrated that casting combined with regular physiotherapy can improve not only joint ROM, but also the quality of the functioning of the affected limb.¹⁴ Furthermore, it has been shown that casting can be effective in order to improve the effects of botulinum toxin.¹⁵

Casting and the other interventions mentioned (splints and orthoses, surgery, and spasticity reducing medications) can be used in conjunction with stretching.

In the following sections we will review the neurophysiological effects, the effects of viscoelastical properties, stiffness and ROM, and treatment and prevention of contractures.

Neurophysiological effects

The effects of stretching on spasticity may be explained by a change in the excitability of motoneu-

rons supplying the spastic muscle. A few studies have examined the changes in motoneuronal excitability after stretching, in patients with SMD. Three studies reported positive effects of stretching,¹⁶⁻¹⁸ while one study did not report any significant effect.¹¹ Rochester *et al.*¹⁶ compared the effects of a program consisting of eccentric contraction plus muscle stretch to eccentric contraction alone, in healthy subjects and patients affected from a neurological disorder which caused ankle spasticity. The neurological patients who received the eccentric contractions showed a significant and maintained increase in the Hoffmann reflex (H-reflex) when compared to the healthy subjects.¹⁶ On the contrary, neurological patients showed a non-significant decrease in the mean amplitude of H-reflexes after receiving the eccentric contraction plus muscle stretch.¹⁶ Authors concluded that the application of a stretch following eccentric contractions decreased motoneuron excitability and may thus be beneficial to decrease spasticity whilst strengthening the muscle.¹⁶ A second study by Suzuki *et al.*¹⁷ investigated the excitability of spinal neural function during stretching exercises in patients with cerebrovascular disease. H-reflex was analyzed before, during, and after one minute of continued stretching of the affected arm.¹⁷ H-reflex was recorded from the abductor pollicis brevis on the affected side after stimulation of the median nerve in the supine position.¹⁷ In patients with moderately increased muscle tonus, the persistence, amplitude and amplitude ratio of H/M recorded during stretching was lower than those recorded before and after.¹⁷ In patients with slightly and markedly increased tonus, H-reflex was the same before, during, and after continued stretching.¹⁷ It was suggested that excitability of spinal neural function during one minute of continued stretching was inhibited in the patients with moderately increased muscle tonus caused by cerebrovascular disease.¹⁷ Al-Zamil *et al.*¹⁸ tested the effects of sustained continuous mechanical stretch on elbow flexors spasticity. Sixteen patients with mild to moderate spasticity of the elbow joint were studied. Reduction of spasticity was measured as a decrease in the amplitude of the EMG response to the passive stretch.¹⁸ Mechanical stretch was maintained by applying a five pound sand bag to the anterior proximal part of the wrist joint for thirty minutes. EMG recordings were made before and after a continuous stretch of the elbow flexors at the end of ten, twenty, and thirty minute intervals

throughout the mechanical stretch. In all patients, a marked decrease in the amplitude of the EMG response (average 82%), as well as a higher threshold to the passive stretch response, was obtained after the mechanical stretch. The effect was maintained and observed by the patients for up to two to three hours.¹⁸

Bakheit *et al.*¹¹ examined the effect of isotonic (with and without weight bearing) and isokinetic muscle stretch on the excitability of the spinal alpha motor neurones (aMN) in patients with post-stroke spasticity and in healthy control subjects. A single 20-min session of isotonic muscle stretch (with or without weight bearing) or isokinetic stretch was delivered to the ankle plantar flexors. The effect of these types of muscle stretches on the excitability of aMN was assessed by measuring the latency of the H-reflex and the ratio of the amplitude of the maximum H-reflex (Hmax) to that of the maximum action motor potential of the soleus muscle (Mmax). The Hmax:Mmax ratio was significantly higher in patients with spasticity than in healthy control subjects. However, there were no statistically significant differences in the H-reflex latency or the change in the Hmax:Mmax ratio between the baseline values and those recorded immediately after the therapy intervention or 24 hours later for each type of muscle stretch. Similarly, there were no significant differences in these variables between the interventions. Thus, neither isotonic muscle stretch (with or without weight bearing) nor isokinetic stretch had a statistically significant effect on the excitability of the aMN in patients with muscle spasticity. This suggests that the previously reported reduction in spasticity after muscle stretch is due to mechanisms other than the direct effect on aMN.¹¹

In summary, the literature examining the effect of stretching on motoneuronal excitability is very scant. Some studies suggest that stretching may induce a reduction of motoneuronal excitability. However, these results are still not sufficient to clearly substantiate this hypothesis.

Effects on viscoelastical properties, stiffness and ROM

Elastic property refers to the capability of the muscle-tendon unit to return to its original length after being stretched. In general, an elastic structure immediately returns to its original length after the

stretch is released. However, this does not occur with muscles because of their viscous properties, which explains why muscles stretch slowly when placed under stress and return to their original length slowly when the stress is removed. However, if a stretch is sustained for a prolonged time, or if there is insufficient recovery before a new stretch, the muscle-tendon unit does not return to its original length. In addition, the muscle will continue to stretch over a finite period of time even if the load is the same. Muscle stiffness is equal to the change in length that occurs, divided by the force applied, and changes in the viscoelastic properties of the muscle-tendon unit due to stretches may explain gains in ROM.¹⁹

Some studies investigating the effect of stretching on muscle viscoelastic properties, stiffness and ROM in patients with spasticity are available in the literature. Most of these studies have methodological limitations, such as small sample size, non controlled studies and diversity in methodology (population, interventions and outcome measures).⁶ Thus, in the following section we will concentrate on randomized controlled trials (RCT).

Only two studies focused on stretching procedures in upper limb spasticity. In the first, Carey²⁰ evaluated the effects of a manual stretch of extrinsic finger flexor muscles on finger extension movement control and force control in 16 patients with spastic hemiparesis. Results showed that the manual stretch treatment could be effective in improving the control of the active finger extension movement at the metacarpophalangeal joint within the available range of active movement, while no significant effects were reported regarding the isometric finger extension force applied during the movement. No follow-up evaluation session was performed, making it impossible to know if this effect could be maintained in the short- or long term.²⁰ In the second study, De Jong *et al.*²¹ investigated the effectiveness of a positioning procedure for the hemiplegic arm in 19 patients with subacute stroke. All patients underwent conventional rehabilitation care. Nine of them additionally received a positioning procedure for two 30-min sessions a day, five days a week, for five weeks. The upper limb positioning procedure consisted of positioning the arm in a specific elongation posture (*i.e.*: shoulder abduction, external rotation, elbow extension and forearm supination). Passive ROM of five arm movements using a hydrogoniometer, as well as resistance to passive movement at the el-

bow using the Ashworth Scale were assessed before treatment and at 5 and 10 weeks after treatment by two blinded assessors. Results showed no significant treatment effects in the passive ROM.

In contrast to upper limb spasticity, studies regarding both instrumental and non instrumental procedures are available for lower limb spasticity. Maynard *et al.*²² found that a single 20-minute session of isokinetic or isotonic muscle stretch (with or without weight-bearing) of the ankle plantar flexors had no clinically observable effect on the gait of hemiplegic stroke patients as assessed by means of kinematic, kinetic and spatio-temporal analysis. Brar *et al.*²³ enrolled 30 patients with multiple sclerosis with minimal to moderate spasticity. Patients were randomized in four groups to evaluate the effects on MS-related spasticity of baclofen alone, stretching regimen with placebo, placebo alone, and stretching regimen with baclofen. Resistance against passive knee flexion was measured by means of a Cybex II isokinetic unit and changes in spasticity were evaluated with the Ashworth scale. Results showed that treatment with baclofen alone significantly improved moderate quadriceps spasticity. A trend indicative of enhancing the beneficial effects of baclofen was noted when stretching exercises were added to the pharmacological treatment. Bressel and McNair²⁴ compared the effect of prolonged static versus cyclic calf stretching on passive ankle joint stiffness, torque relaxation, and gait in people with stroke. Results showed that ankle joint stiffness decreased after both prolonged static (35%) and cyclic stretches (30%). With regards to torque relaxation, a greater effect was reported after static stretching than after cyclic stretching. On the other hand, gait performance did not appear to be influenced by any of the stretching techniques used. Yeh *et al.*²⁵ compared the effectiveness of constant-torque prolonged muscle stretching (PMS) treatment with constant-angle PMS treatment in the inhibition of ankle hypertonia in patients with stroke. Ankle plantar flexors were stretched using a motor-driven stretching device capable of operating in either a constant-angle or a constant-torque mode for 30 minutes of treatment. Both constant-angle and constant-torque PMS treatments significantly reduced spasticity (evaluated by means of the Modified Ashworth Scale) and improved the ankle's ROM. Both approaches reduced the viscoelastic components of the ankle joint muscles, but the degree of change in these muscles was more

evident in patients who underwent constant-torque PMS than in those treated with constant-angle PMS. Hale *et al.*²⁶ aimed to investigate which duration of PMS, two, ten or thirty minutes, was optimal in reducing spasticity in spastic quadriceps femoris muscles in adult patients following traumatic brain injury. Twenty-nine spastic muscles were stretched for the three predetermined durations. Results showed that the most beneficial duration of PMS in decreasing spasticity was ten minutes. Harvey *et al.*²⁷ performed an assessor-blinded randomized controlled trial in order to determine the effect of 4 weeks of 30 minute daily stretching on ankle mobility in 14 patients with a recent spinal cord injury. Treated ankles were stretched continuously into dorsiflexion with a torque of 7.5 N x m each weekday while the contralateral ankles received no stretches. Torque-angle measurements were obtained with the knee extended and flexed and they were collected before treatment and at 2, 4, and 5 weeks after the onset of the treatment. Results showed that the stretching intervention did not significantly change the ankle passive torque-angle curves at any time.

Treatment and prevention of contractures

Joint contractures are a common problem that impedes the rehabilitation of patients following traumatic brain injury, cerebral palsy, multiple sclerosis and stroke. It is defined as an abnormal shortening of muscle tissue, rendering the muscle highly resistant to passive stretching and may result in joint distortions or deformities. Children with cerebral palsy, spina bifida and arthrogryposis have progressive contractures that inhibit function and limit their development. In this section, we will explain the effect of passive stretching on contracture development.

Contractures can be prevented by the maintenance or increase in numbers of sarcomeres in series, together with the maintenance of tendon length and connective tissue elasticity. Sarcomere numbers are maintained by stretching when the muscle is held in a lengthened position. Immobilisation in a lengthened position maintains that muscle length but may cause loss of sarcomeres from its antagonist and risks connective tissue accumulation and loss of elasticity. Connective tissue does not appear to accumulate in the presence of contractile activity. Plastic deformation of connective tissue is achieved by prolonged low force stretching, and enhanced

when connective tissue is warmed prior to stretching and cooled prior to release of stretch. Contractures require long-term management when certain conditions occur which cause progression of/or re-occurrence of contractures.²⁸

Studies dealing with the effects of stretching in contracture development are scant.^{14, 21, 29} It is interesting to report the study by Tardieu *et al.*,²⁹ who monitored the amount of time a spastic muscle was in a stretched position during the execution of activities of daily living. They found that contractures did not occur when muscles were in a lengthened position for more than six hours per day.

In summary, to prevent contracture development, stretching of the muscles should be prolonged for various hours during the day mainly applying the casting technique.¹⁴

Muscle strengthening

Muscle weakness, defined as the inability of a patient to generate normal levels of muscle force under a specific set of testing conditions,³⁰ can be a major factor contributing to disability in patients with upper motor neuron syndrome, suffering from SMD.³¹ Previous studies have described that the mean degree of strength in the involved limb varied from 23% to 94 % of that on the ipsilesional side in patients with stroke.³² Moreover, the amount of weakness has been reported to correspond to the severity of the brain lesion and to be more pronounced distally (*i.e.* mainly involving ankle plantar flexors at the lower limb) than proximally (*i.e.* hip flexors at the lower limb).³² From an epidemiological point of view, it is interesting to note that data about spasticity distribution are similar to data regarding weakness.³³

With regards to the physiopathological mechanisms underlying the loss of strength after an upper motor neuron lesion, previous literature has described that immediately after the onset, reduced force production is due to a loss of descending input to spinal motor neurons with a lower activation of motor units.³⁴ On the other hand, during the chronic phase of illness, reduced force production is due to a decrease of cross sectional area of muscle;³⁵ reduction of motor units as a consequence of disuse;³⁶ muscle fiber atrophy and contracture; changes in the spatial and temporal patterns of muscle acti-

vation (*i.e.* coactivation) causing an inefficient EMG-torque relationship; loss of functional motor units and changes in the properties of remaining units; limited voluntary ROM in agonists with muscles producing decreased maximal force due to activation on a suboptimal portion of the force-length relationship.³⁰

Thus far, three main assumptions regarding the relationship between muscle spasticity and muscle power have greatly influenced the rehabilitation procedures of patients with spastic movement disorder. The first one upholds that spastic muscles are also strong muscles. Based on this hypothesis, a strong spastic agonist would result in a weak antagonist with a consequent muscle imbalance imposed by strong spastic muscles.^{33, 37-39} The second assumption sustains that spastic muscles are weak muscles and that the amount of weakness is related to the amount of spasticity and severity of brain lesion.^{33, 40} The third supposition sustains that although no strict relationship exists between spasticity and muscle weakness, loss of strength and spasticity could coexist. The muscle would be weak because movement under pathological patterns requires the muscles to use as much energy as they have available, leading to premature muscle overtiredness.^{33, 41, 42} These considerations are very relevant from a rehabilitative point of view because embracing one hypothesis over the other could completely change our treatment approach. For example, based on the first assumption (spastic muscles are strong muscles), strengthening of spastic limbs has been traditionally considered useless and also contraindicated. This is clearly reported by Bobath, who stated that "heavy resistance training should be avoided in patients with upper motor neuron lesions (because) in spastic conditions, if disinhibited, the use of effort, irradiation, mass patterns and especially tonic reflexes to strengthen muscles, will only reinforce the few existing abnormally increased reflexes and, with it, increase spasticity".⁴³

This supposition (strength exercises increase spasticity) has not been confirmed by several recent studies.^{30, 31, 34} In 2004, Morris *et al.*³¹ examined eight articles reporting data about the improvements in activity limitation, functional impairment and participation restriction after progressive resistance strength training in patients with stroke. With regards to the relationship between spasticity and strengthening intervention, they stated that no evidence shows

that strength training increases spasticity or reduces articular range of movement at the affected limbs.³¹ Moreover, authors reported that patients were shown to significantly reduce musculoskeletal impairment after resistance strength training.³¹ Ada *et al.*³⁴ investigated, through a systematic review of the literature with meta-analysis of randomized trials, if strengthening interventions would increase strength, spasticity and activity in patients with stroke. Their results confirmed not only that strengthening interventions are not harmful in patients with spasticity, but also that augmented muscle strength improves functional profile after stroke.³⁴ According to these findings, authors suggested that the fear of worsening a patient's spasticity is not a reason to avoid strength training, which should, instead, be part of rehabilitation programs for patients suffering from SMD, especially in the first six months following a stroke brain lesion.³⁴ Pak and Patten³⁰ also confirmed that strengthening does not exacerbate spasticity during or after a course of treatment. Moreover, they provided evidence about the effectiveness of strengthening interventions for improving functional outcome and quality of life in patients with upper neuron lesions.³⁰

On the basis of these considerations, several rehabilitative approaches has been proposed in order to improve muscle strength in patients with upper motor neuron syndrome, such as muscle training, biofeedback and electrical stimulation (see physical modalities section, this article).

Muscle training

With regard to muscle training, different approaches aimed at improving resistance have been reported to be effective in patients with upper motor neuron syndrome.^{30, 31, 34} Previous studies have reported that Progressive Resistance Strength Training (PRST) is one of the most common therapeutical approaches in these patients.^{31, 34} The PRST refers to progressive increases in resistance to a muscle as training induces greater ability to produce and sustain force.³¹ One of the key elements of PRST is to provide sufficient resistance by completing a relatively small number of consecutive repetitions (usually less than 12) before fatigue. Moreover, during the training, the amount of load should progressively increase as strength increases. Finally, the programme should continue for a sufficient duration (a minimum of four

weeks) for benefits to accrue.^{31, 44} Another common approach is muscle re-education.^{34, 45} It is defined as a strengthening intervention that progresses from passive and/or assisted movements to active and resisted movements, using manual resistance, weights, isokinetic machines and circuit-training.^{30, 34, 45} In regards to neurorehabilitation practice, Pak and Patten³⁰ reported that there is currently no gold standard to guide the development of strengthening protocols in patients with SMD. However, based on the clinical studies reviewed, they recommended some basic parameters for resistance training. In particular, they suggested that: 1) the level of resistance should be a one-repetition maximum at 60%-80%, reassessed every 2 weeks; 2) the number of repetitions per set should not exceed 12 and each set (minimum 3 per session) should be composed of 8 to 10 exercises; 3) the training should be conducted 3 times per week for a minimum period of 6 to 12 weeks; 4) functional task-specific activities should be incorporated. Moreover, they recommended to avoid strength training in: 1) non-neurologically stable patients; 2) post-surgical patients; 3) patients with severe osteoporosis or acute joint injuries; 4) patients with haemophilia or other blood disorders; 5) patients with severely limited ROM.³⁰

Biofeedback

Biofeedback (BFB) is a therapeutical approach for muscle strengthening that allows subjects to gain conscious control over a voluntary but latent neuronal function by alerting them, with an auditory or visual cue, that their efforts have activated a targeted muscle together with a specific neuromuscular pathway.⁴⁶ According to a recent Cochrane review,⁴⁷ it is not possible to definitively state its effectiveness for enhancing functional recovery in patients with upper motor neuron syndrome. However, some studies suggested that BFB combined with standard physiotherapy could produce improvements in muscle strength, functional recovery and gait quality compared to standard physiotherapy alone.⁴⁷ With regards to the effects of BFB on patients with SMD, Lourenção *et al.*⁴⁸ reported that BFB (combined with functional electrical stimulation and occupational therapy) had some positive effects on joint ROM and on the recovery of upper limb function. Moreover, they reported not only that the degree of spasticity does not increase after treatment, but also

that patients who underwent BFB plus functional electrical stimulation and occupational therapy presented a reduction in spasticity greater than patients who performed functional electrical stimulation and occupational therapy alone. These findings were explained arguing that BFB training involves both skilled repetitive movements and inhibition of unwanted activity of antagonistic muscles.⁴⁸

Physical modalities

Several studies have reported the usefulness of physical agents in treatment of muscle spasticity. The effects of different physical modalities such as shock wave therapy, ultrasound therapy, cryotherapy, thermotherapy, vibration and electrical stimulation, have been examined.

Shock wave therapy

Shock waves are defined as a sequence of single sonic pulses characterized by high peak pressure (100 MPa), fast pressure rise (<10 μ s) and short duration (10 μ s).⁴⁹ Their efficacy in the treatment of bone and tendon diseases such as tendonitis calcarea of the shoulder,^{50, 51} pseudoarthrosis,^{49, 52, 53} epicondylitis⁵⁴ and plantar fasciitis⁵⁵ has been demonstrated. After the successful application of shock waves in the treatment of muscular contractions of athletes, two recent papers examined their efficacy on spasticity in adult and paediatric patients.^{49, 56} With regards to adult patients, Manganotti and Amelio⁴⁹ reported that a single treatment of shock wave therapy focused on flexor hypertonic muscles of the forearm and the interosseus muscles of the hand in patients with stroke resulted in a significant reduction of muscle tone that lasted for over three months. In regards to paediatric patients, the same researchers described that a single session of shock wave therapy focused on plantar flexors in children affected from cerebral palsy with spastic equinovarus foot produces a significant long-lasting (>12 weeks) reduction in muscle tone.⁵⁶ Even if the therapeutic mechanism of shock wave therapy on spastic muscles still remains unclear, the authors suggested that a direct effect on fibrosis and rheologic components of the hypertonic muscles has to be considered, in accordance with the therapeutic effects documented on bone and tendon disease.⁵⁰⁻⁵⁵ In addition, Amelio

and Manganotti^{49, 56} proposed that another possible explanation of the shock waves therapeutical effect in spasticity could be related to shock waves induction of the enzymatic nitric oxide synthesis that would be involved not only in neuromuscular junction formation in the peripheral nervous system but also in important physiological functions of the central nervous system, such as neurotransmission and synaptic plasticity.

Ultrasound therapy

Ultrasound therapy is assumed to have thermal and mechanical effects on the target tissues resulting in an increased local metabolism, circulation, extensibility of connective tissue and tissue regeneration with improvements in pain syndromes, swelling and articular ROM.^{57, 58} With regards to muscle effects, ultrasounds combined with static stretch showed to significantly improve the muscle extensibility compared to static stretch only, in healthy people.⁵⁹ Ansari *et al.*^{57, 58} published two interesting papers about the effect of continuous ultrasounds in reducing spasticity on small samples of patients. In particular, they reported that fifteen 10-minute sessions of continuous ultrasound therapy over a 5-week period (frequency 1 MHz; intensity 1.5 W/cm²) significantly reduced alpha motoneuron excitability (as measured by Hmax/Mmax ratio) and ankle plantar flexors spasticity (as measured by the Ashworth score) in patients with stroke.^{57, 58} Authors justified their findings suggesting that ultrasound therapy not only allows viscoelastic changes in spastic muscles but also decreases the sensitivity of the muscle spindle to stretch and alpha motoneuron excitability by increasing the tissue temperature.^{57, 58} On the other hand, a recent paper from the same authors compared the efficacy of ultrasound and infrared therapy in the management of spasticity, reporting that neither infrared nor ultrasound therapy reduced electrophysiological and clinical measures of spasticity.⁶⁰

Cryotherapy

Local muscle cooling has been described to temporarily decrease spasticity and clonus mainly by reducing the sensitivity of the muscle spindle to stretch.⁶¹ Furthermore, skin cooling has been suggested to have an antispastic effect by increasing

pain threshold and consequently reducing receptor sensitivity of low-threshold afferents.⁵⁹ Cold can be applied locally by rubbing with ice packs and cubes (not applicable to patients with cold hypersensitivity) or using evaporative sprays such as ethyl chloride (not applicable for longer than 10 minutes).⁵⁹ The average application time is 20 minutes, even if it should be longer in heavier people and adjusted to maintain adequate body temperature, in order to avoid shivering associated with hypertonia increasing.⁵⁹ Moreover, cooling of the upper limbs must be applied with caution, especially in patients with coronary disease, to avoid angina attacks.⁵⁹ The effects of cooling have been described to last for only two hours after treatment.⁵⁹ In regards to the optimal cooling temperature, Lee *et al.*⁶² evaluated the effect of cold air therapy (applied by a machine that uses dry ice to decrease air temperature allowing applications longer than 10 minutes) in relieving spasticity in an animal model. Considering the duration of treatment effect and its safety, authors reported that an intramuscular temperature of 30 °C would be optimal to maintain effects between 30 and 60 minutes after treatment in spinal injured rabbits.⁶² Harlaar *et al.*⁶¹ have reported a similar drop in muscle temperature to effectively reduce spasticity in humans after a local, 20-minute application of cold packs (-12 °C). From a clinical point of view, the local cooling of spastic muscles could be considered a useful and inexpensive tool that could be combined with active training of the antagonist muscles and also used to hinder muscle hypertonia and clonus during casting procedures.

Thermotherapy

Although cooling effects on spasticity have been investigated more than the effects of superficial heating, thermotherapy has been reported to decrease muscle tone, reduce muscle spasms and increase the pain threshold in patients with muscle hypertonia.⁵⁹ In regards to the effects of global heating for spasticity, Matsumoto *et al.*⁶³ described that F-wave amplitude and F-wave/M-response ratio significantly decreased in patients with post-stroke spasticity after a 10 minute warm water (41 °C) bath. Authors suggested that thermotherapy antispastic effect is due not only to a relaxation of muscular and other soft tissues, but also to a decrease in gamma-afferent fibre activity that would

lead to a decrease in impulses from the muscle spindles with a consequent inhibition of impulses to the alpha fibres.⁶³ Other superficial heating modalities were discussed in a recent paper by Lee *et al.*,⁶⁴ who reported that passive stretching (5 repetitions x 30 seconds) with prior local heat treatment (20 minutes, 75 °C hot pack application) significantly increases hamstrings extensibility. They hypothesized that hot temperature could have reduced the response of muscle spindles to stretch, rendering the muscle more extensible under passive stretching.⁶⁴ Unfortunately, to the best of our knowledge, no previous study has evaluated the long term impact of heat application on spasticity: this should be taken into account when programming future studies investigating heat effects on spastic hypertonia.

Vibration

Vibratory stimulation has also been found to have anti-spastic effects. Noma *et al.*⁶⁵ recently reported the effectiveness of direct application of vibratory stimuli to upper limb spastic muscles of the hand, forearm and upper-arm in patients with stroke. Authors described that patients reached significant short-term (30 minutes) improvements in muscle tone (Ashworth score), electromyographic (F-wave) and motor function (finger tapping, active ROM, test for hand function) parameters after treatment.⁶⁵ In addition, the use of vibrating platforms for whole body vibration training demonstrated not only to reduce muscle tone (especially in the knee extensors), but also to increase gross motor function in patients with spasticity.^{66, 67} In order to explain these results, authors suggested that muscle strengthening due to whole body vibration training could play an important role in the functional improvement seen in their studies.⁶⁷ Finally, an interesting issue regarding vibration therapy in patients with spasticity is the anti-spastic role of penile vibratory stimulation in men with spinal cord injury (SCI).^{68, 69} This therapeutic approach for anejaculation showed to significantly reduce lower limb muscle tone (measured for several hours after application).^{68, 69} Authors hypothesized that the activation of pudendal afferents after penile vibratory stimulation could influence the neuronal circuits in the lumbar spinal cord involved in the pathophysiology of lower limb spasticity.⁶⁹

Electrical stimulation

In regards to the application of electrotherapy for treatment of spasticity, Transcutaneous Electrical Nerve Stimulation (TENS) applied on the common peroneal nerve, spinal dermatomes or over the region of the spastic muscles has been reported to reduce muscle tone in patients with stroke, spinal cord injuries and cerebral palsy.^{59, 70-75} TENS anti-spastic effect has been hypothesized to be related with the production of β -endorphins which may decrease the excitability of the motor neurons, and based on the gate control theory, cause a reduction in nociceptive inputs.^{71, 72} Moreover, TENS has been suggested to facilitate cortical synaptic reorganization and motor output by increasing sensory input due to larger diameter A _{α} , β fibre stimulation.^{71, 72} Clinical effects of TENS in patients with stroke have been investigated by Ng *et al.*⁷⁰ who showed that TENS can increase the effectiveness of a task-related training with regards to both spasticity and walking ability measures. As to the effectiveness of TENS on spasticity in patients with multiple sclerosis, controversial evidence was found.^{76, 77} Although a pilot study by Armutlu *et al.*⁷⁶ reported that high-frequency TENS applied to the spinal dermatomes significantly decreases Ashworth scores and myoelectric activity in patients with multiple sclerosis, a more recent trial by Miller *et al.*⁷⁷ on a larger sample of patients found that TENS has no statistically significant effects on lower limb spasticity, while a reduction of pain and muscle spasms was found only in the case of prolonged TENS application (8 hours per day). Thus, the effectiveness of TENS for treatment of spasticity in people with multiple sclerosis needs to be further evaluated by means of randomized controlled trials.

Electrical stimulation leading to muscle contraction (such as rectangular waves or Faradic stimulation) has also been used for the treatment of spasticity. Its antispastic effect has been suggested to be based on mechanisms of facilitating Renshaw cell recurrent inhibition,⁷⁸ antagonist reciprocal inhibition,⁷⁸ cutaneous sensory habituation⁷⁸⁻⁸⁰ and augmentation of Ib fibres activation.⁷⁸ Furthermore, electrical stimulation showed to enhance the effects of neurorehabilitation inhibitory techniques (*i.e.* Bobath approach).⁸¹ The principal advantages of electrical stimulation are the possibility to modulate the intensity of intervention and local appli-

cation, with a consequent opportunity to modulate the therapeutic effect.^{78, 81} Concerning the methods of stimulation, van der Salm *et al.*⁸² compared the effectiveness of antagonist, agonist and dermatome stimulation in triceps surae spasticity in patients with spinal cord injury. Agonist stimulation led to significant improvement in Ashworth scores, while antagonist stimulation led to an increase of stretch reflex-initiating angle.⁸² Authors suggested that the effects of agonist stimulation could be primarily due to a reduction of muscle stiffness as a consequence of visco-elastic muscle properties modification.⁸² On the other hand, antagonist stimulation would probably be more effective in reducing spasticity by modulating muscle spindles activity.⁸²

Pain and spasticity

Pain is one of the most frequent symptoms in rehabilitation, particularly in the field of neuromotor rehabilitation. The International Association for the Study of Pain (1986) defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, and could be considered as the perception of an aversive or unpleasant sensation involving both abstraction and elaboration of sensory inputs.⁸³ This description emphasizes that pain is different from nociception, which refers to the reception of signals in the central nervous system (CNS) evoked by activation of different nociceptors, in order to provide information on tissue damage. The need for this distinction arises from the recognition that pain is a multidimensional phenomenon involving both an objective (the physiological tissue damage causing the pain) and subjective (perceptual, affective, cognition and behavioural component) dimension.⁸⁴

Although pain has been grouped into different categories referring to duration (acute and chronic pain) and type (neuropathic, nociceptive and psychogenic pain), a clear classification is not always possible. Frequently, different problems in rehabilitation can cause pain, and many patients show "mixed" pains that do not fit into either category.⁸⁴

Patients with CNS lesions (such as spinal cord injury,⁸⁵ stroke,⁸⁶ brain injury,⁸⁷ multiple sclerosis,⁸⁸ etc.) frequently report mixed clinical features' associated with different neurological symptoms. The

prevalence of pain in upper motor neuron syndrome differs among the different diseases, ranging from 19% to 74% of patients with stroke,⁸⁶ 60-70% of patients with spinal cord injury,⁸⁹ 22-95% of patients with traumatic brain injury⁸⁷ and more than 40% of patients with multiple sclerosis.⁹⁰

In particular, pain is frequently associated with spasticity, but often neglected in articles dealing with pain. Pain leads to the worsening of a physical disability,⁸⁹ causes a limitation of activity and participation,⁸⁵ and increases spasticity, creating a spiralling course of more pain and disability.⁹¹

Pathophysiology of pain in spasticity

The pathophysiology of pain in spasticity is not well understood.⁹¹ Ward and Kadies⁹¹ point out that spasticity is a part of the upper motor neuron syndrome involving both hypertonia and peripheral secondary structural changes of muscles, including alteration in tendon compliance and changes in the muscles fibres themselves. The clinical effects of spasticity, thus, could be summarized in the interplay between neurological impairment and biomechanical changes. This combination could give rise to pain and the following three main clinical manifestations can be described: mobility and posture abnormalities, tonic spasms and cortical pain.⁹¹

As to mobility and posture abnormalities, SMD causes aberrant biomechanical forces on limbs and the trunk. It then causes abnormal postures characterized by an imbalance in muscle contractions, leading to a progressive loss of active and/or passive ROM. A typical example would be the appearance of shoulder pain in patients with upper limb spasticity, in which pain could be due to a pathological structural misalignment in joints and the skeleton. Thus, soft tissue damage (*i.e.* muscles shortening, tendinopathies etc) and joint pathology (*i.e.* adhesive capsulitis) can be a cause of pain. On the other hand, the abnormal muscle contraction and the tonic muscle activation could lead to a stamping out of muscle vessels, reducing the oxygen availability. This is a very complex situation because the muscles' overactivity most likely causes the level of oxygen consumption to be larger than normal. Hypoxia leads to the release of inflammatory substances and then to the activation of muscles nociceptive receptors, increasing pain.⁹² Furthermore, pain increases spasticity, once

again creating a spiralling course of more pain and disability.

With regards to tonic spasms, the upper motor neuron syndrome can produce dystonic postures and muscles spasms that by themselves can generate pain. Central pain syndrome is another symptom that frequently affects patients with CNS damage.⁸⁷ It is thought to be generated mainly by a disturbance of thalamo-cortical transmission or lesions in the ascending nociceptive pathways with consequent widespread sensory disturbances.⁸³ The resulting dysesthesiae and paraesthesiae can be quite distressing and lead to an indirect increase in muscle tension and spasticity.

Clinical features

The pain associated with spasticity can be characterized by prolonged symptoms due to postural problems and can be defined as follows:⁹¹

- sharp in the case of muscle spasms
- occurring anywhere, even if it is typically worst in the proximal limb and trunk
- perceived during the rest position and increases during active and passive movement of the body part
- worst when musculoskeletal abnormalities occur and usually does not tend to radiate.
- some of the features of associated arm and hand pain may be linked to more proximal problems in the neck and shoulder.

Management

The focus of treating patients with pain due to spasticity is the treatment of the underlying condition, as the two are co-related.⁹¹ Furthermore, pain relief in this type of condition requires a multi-professional approach and a comprehensive treatment plan consisting of different physical and medical procedures.⁹²

The first treatment aim is to decrease nociceptive stimuli, followed by improving functional physical activity (when possible), and providing cognitive and behavioural strategies to assist in resuming (when possible) daily life activities.⁸⁴

Essentially two main approaches in pain management associated with spasticity can be identified: a pain centred approach and a spasticity-centred approach.

Pain centred approach

This consists of medical (*i.e.* anti-inflammatory drugs, antidepressant, opioid and non-opioid drugs, anticonvulsant and Cannabis resin) and physical interventions^{84, 91, 93} (*ie* heat/cold therapy, TENS, electrotherapy, hydrotherapy and brain stimulation procedures) aimed at reducing pain at a specific body part where it is perceived. This approach could be effective in order to gain a better control of the pain. Therefore, controlling the pain may be fairly straightforward, and the pain relief may in itself contribute to a decrease of muscle tone.

Analgesic medication has been recommended only for a short-term period, adjunct in the overall treatment program,⁸⁴ because the most important way to relieve pain is to control the underlying spasticity.⁹¹

Spasticity centred approach

This consists of medical and physical interventions focused on decreasing spasticity and painful spasms (defined as cramping, pulling pain, more commonly involving the lower limbs and mainly occurring during the night), with a consequent improvement in limb posture and joint ROM. In particular, painful spasms are a frequent symptom in patients with spinal cord injuries and multiple sclerosis.⁹⁴ They show a sudden-onset, characterized by the appearance of dystonic posturing, which can involve uni- or bilaterally, with a stereotyped pattern.⁹⁵ The attacks are usually brief, lasting less than 2 minutes, and pain is usually localized in the same area as motor symptoms.⁹⁵ Studies have suggested that painful tonic spasms may be caused by both an exaggerated activation of axons within a partially demyelinated lesion⁹⁶ and an axonal irritation secondary to the release of excitatory soluble factors during inflammation.⁹⁵

Medical interventions can be categorized in generalized (oral administration of baclofen, tizanidine, dantrolene sodium, diazepam), regional (intrathecal baclofen) and focal (botulinum toxin and phenol blockade) spasticity treatment. Baclofen is probably the antispastic drug of choice in spinal cord spasticity^{89, 97} and it has been effective not only at improving urinary and bladder control, but also at reducing reflex spasms at the Penn Spasm Scale.^{97, 98}

Carbamazepine has been shown to be potentially effective in reducing painful tonic spasms, probably by binding to sodium channels and then limiting the occurrence of firing action potentials. It is commonly used for central neuropathic pain in multiple sclerosis, although often with inadequate results.⁹⁴ Other oral agents such as dantrolene sodium, diazepam and tizanidine have been reported to be effective for controlling spasticity and pain symptoms following upper motor neuron lesions, even if adverse effects are commonly reported.^{91, 97}

Focal spasticity treatment has also been proved to be very effective in reducing pain co-related symptoms. These positive effects have been reported mainly about botulinum toxin (that acts at the neuromuscular junction by reducing the release of acetylcholine, and thus decreasing muscular contraction)^{99, 100} treatment, while phenol and alcohol injections, when injected into a mixed nerve, may cause pain, dysesthesia and causalgia.¹⁰¹

Shoulder pain in particular has been reported to be one of the most common problems after stroke^{86, 102-104} and it is an important contributor to length of hospital stay,¹⁰⁵ mood disturbances appearance,¹⁰⁶ and decreased quality of life.¹⁰⁷

The common shoulder movement pattern observed in patients with spastic hemiplegia is primarily adduction and internal rotation, with a consequently limited active and passive ROM of the shoulder.^{91, 108}

Spasticity of the subscapularis muscle and of the pectoralis major limits the abduction, external rotation and flexion of the shoulder, and aged studies showed that the surgical release of these muscles could improve both the shoulder's ROM and shoulder pain after CNS lesions.

Research data points out the role of spasticity in hemiplegic shoulder pain and suggests the beneficial role of botulinum toxin injection into the subscapularis muscle^{103, 109, 110} and pectoralis major¹¹¹ in reducing shoulder pain. It is interesting to note that the relief of pain often occurs before the decrease in muscle contractions, suggesting that botulinum toxin could have more complex mechanisms of action than hypothesized.¹¹² Studies have demonstrated that botulinum toxin could inhibit the release of substance P and other neuromodulators.¹¹³ Therefore, botulinum toxin is becoming an effective and innovative approach for controlling pain, in particular in conditions such as headache, myofascial pain,

cervical dystonia and hemiplegic shoulder pain.^{109, 110, 114-119}

It is important to note that botulinum toxin has to be considered a therapeutic tool which should be coupled with specific physical treatment, such as physiotherapy aimed at improving posture and positioning, stretching of particularly spastic muscles, strengthening antagonist muscles and neuromotor rehabilitation. Furthermore, casting and splint positioning could be helpful in order to reduce limb posture abnormalities and maintain muscle lengthening.¹⁵

Conclusions

The present paper showed that although procedures for treatment of SMD are predominantly based on pharmacological treatment, a large number of studies dealing with non pharmacological (rehabilitation) treatment are available in the literature. As a whole, those studies have important limitations, in that they show great diversity at the levels of methodology, population, intervention, and outcome measures, making a meta-analysis not feasible.

However, these preliminary researches have often shown very promising results. Furthermore, taking into account that each rehabilitation approach could be directed at treating one or more components of the SMD (see: stretching for hindering changes in muscle mechanical properties, muscle reinforcement in order to prevent muscle weakness and mechanical changes, etc.), a treatment protocol combining pharmacological and non-pharmacological interventions could be proposed. In light of the complexity of the SMD condition, a combined treatment approach would probably be more effective than the pharmacological treatment alone.

Future research in this field should focus on selecting the most appropriate rehabilitation procedure for each SMD component, proposing a combined treatment approach feasible in clinical practice, and testing the effectiveness of the approach compared to pharmacological treatment alone, using a RCT design. Future studies should also use similar procedures of intervention and outcome measures.

To conclude, research on the effectiveness of combined treatment protocols in the management of SMD is a very promising field of neurological rehabilitation, which is recommended for the future.

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